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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—10TH YEAR. SYDNEY: SATURDAY, AUGUST 11, 1923. No. 6.

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ACHLORHYDRIA: AN INVESTIGATION INTO ITS NATURE WITH SUGGESTIONS FOR ITS TREATMENT.

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A RECENT study of cases of achlorhydria or absence of free hydrochloric acid from all the samples of gastric contents obtained from patients investigated by the fractional test-meal method, has revealed some most interesting facts which may have a profound influence on the treatment of this condition.

Two curious facts were noticed concerning a number of these patients examined by me at the Melbourne Hospital. When the series of tubes containing the samples withdrawn at quarter-hour intervals were examined, the following was found.

(1) That the condition of patients from whose gastric contents free hydrochloric acid was absent, could be divided into two classes: (*a*) those in which there was little or no clear fluid above the sedimented food after standing for some hours, and (*b*) those in which there was relatively a considerable quantity of fluid. In the former condition the secretion of gastric juice appears to be absent or depressed. In the latter there is a strong suggestion that much alkali has regurgitated from the duodenum and has neutralized the acid entirely, so that what at first sight appears to be a case of non-secretion of hydrochloric acid may really be a condition in which secretion has taken place, but for some reason alkali has been driven backwards from the duodenum into the stomach and has completely neutralized the acid.

(2) In certain other conditions in which the stomach has emptied rapidly, as shown by the early disappearance of sedimented food from the samples, there was a reappearance of food in the later samples.

These facts suggested to me that, owing possibly to some duodeno-jejunal irritation, an exaggeration of the normal antiperistalsis had occurred, producing a backflow of food into the stomach and that such a mechanism might be responsible for the complete neutralization of acid, if this did occur. That this reflux does occur in normal people, thus regulating gastric acidity, is an established fact.

Again the results of my experiments on normal students who showed all varieties of gastric acidity, strongly suggested that at least certain cases of achlorhydria were of the type suggested above. These experiments are described in a previous paper in this journal.[1] They showed that the different

types of gastric acidity—hyperchlorhydria, hypochlorhydria and normal acidity—were dependent to a large extent, if not wholly, on the amount of alkali reflux into the stomach, so that hypochlorhydria, for instance, was due not so much to lowered gastric secretion, as to greatly increased alkaline reflux from the duodenum. There was also evidence that the amount of reflux depended partly on a low gastric tone in normal persons. That many cases of achlorhydria are due to this same increased intestinal reflux is only an extension of the above experimental results.

In order to investigate this theory a number of my patients who on previous examination were shown to have complete absence of free hydrochloric acid, were sought and re-tested. The gruel given to these patients as the test-meal was absolutely salt-free. The total chlorides in each sample were now estimated by Volhard's method. In the majority of the twenty-one patients examined increasing amounts of chloride were found to be present. This could only have come from neutralized hydrochloric acid. Figure I. illustrates a number of

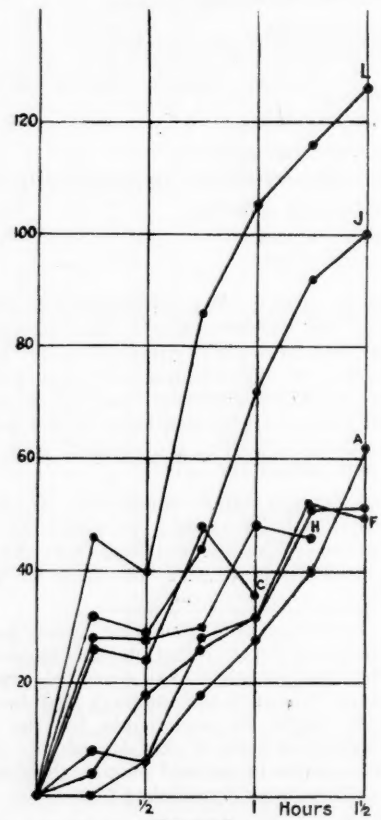

FIGURE I.
Total Chlorides in Test Meal Fractions, from eight cases of apparent achlorhydria.

curves representing the total chloride in each hundred cubic centimetres of gastric juice from patients with apparent achlorhydria. Figure II., for comparison, illustrates similar curves from seven normal persons and patients with hyperchlorhydria

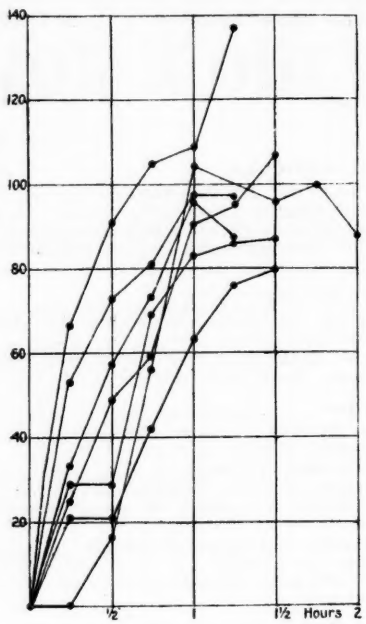

FIGURE II.
Total Chlorides in seven cases of normal acidity and hyperchlorhydria.

and Figures III. (*a*) and (*b*) the curves from patients with pernicious anæmia and *carcinoma gastrica*, in which conditions it was thought that

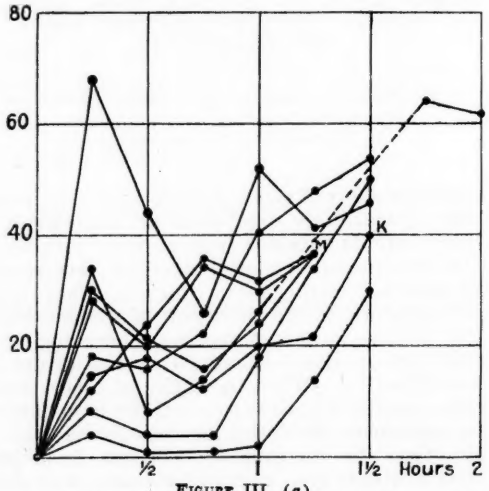

FIGURE III. (*a*).
Total Chlorides in nine cases of pernicious anæmia.

true achylia might exist. In spite of the fact that much dilution with duodenal juices must have occurred, the chloride content of the gastric secretion in the group in Figure I. is sufficient to have given a high gastric acidity in each case, had this

chloride been in the form of unneutralized hydrochloric acid. The fact that the curves are rather lower than those of Figure II. is, of course, due to this dilution by duodenal juices and can be wholly

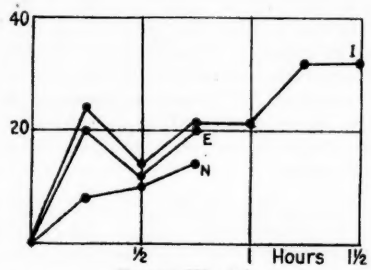

FIGURE III. (*b*).
Total Chlorides in three cases of carcinoma of the stomach.

explained by this fact. Thus it is found that the total chloride curves in those patients with normal or hypernormal acidity reach an average of nearly 100 degrees at one hour, before which time very little dilution with duodenal juices occurs as a rule in

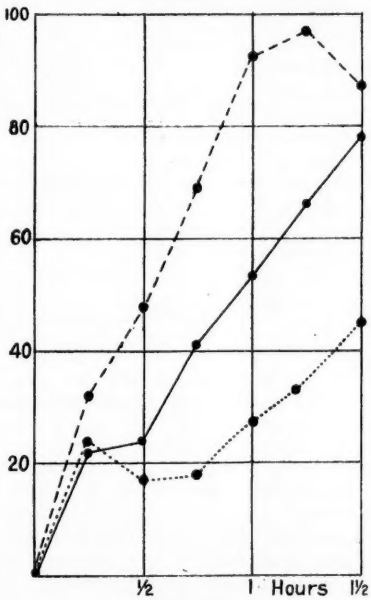

FIGURE IV.
Curves of Average of Total Chlorides: in achlorhydrics (continued line); in normal persons and hyperchlorydrics (interrupted line); in patients with pernicious anæmia (dotted line).

these cases (Figure IV.). Now the strength of acid in pure gastric juice is rather above $^{n}/_{10}$ HCl, while that of alkali in pure pancreatic juice is nearly $^{n}/_{10}$ $NaHCO_3$ and, when the latter is admixed with bile, the alkali strength must be lower than this. If the gastric juice be now neutralized with the mixture of pancreatic juice and bile or even with pancreatic juice alone, it will be seen that a volume of the latter larger than the volume of gastric juice will be required, in order that the total chloride strength of the mixture will be reduced to at least half, *id est* to at least 50 degrees, and generally to less than this. An examination of the total chloride values of the achlorhydria patients in Figure I. shows that this figure approximately represents their average value at one hour, though the presence of food would lessen the amount of this fall in acidity. It may be stated then that the secretion in these apparent achlorhydria patients is as great as that in normal persons and in the hyperchlorhydria patients. These values represent, of course, not the total amount of chloride present in the stomach, but the chloride per hundred cubic centimetres of gastric contents; it is, therefore, the strength of the chloride.

Achlorhydria would appear to be divisible into two classes: (i.) True achlorhydria or achylia, in which the secretion of hydrochloric acid is negligible, as in cancer of the stomach, gastritis and probably some cases of pernicious anæmia and (ii.) false achlorhydria, in which acid secretion occurs in normal amount, but is at once completely neutralized by duodenal alkali reflux. These appear to form the majority of all cases.

In addition to the total chloride estimations a rough determination of the amount of pepsin present was made in each sample of some of the patients. For this purpose Mett's tubes were used. They consist of short lengths (about 1.5 centimetres) of small bore glass tubing filled with coagulated egg-white. One of these was dropped into each sample, to which was also added an equal volume of 0.4% hydrochloric acid and the whole incubated at 37° C. for twenty-four hours. The pepsin of the original gastric juice has, of course, been diluted four times, to at least an equal volume by the intestinal juices and to double this again by the 0.4% acid added. The lengths of coagulated egg-white, measured in millimetres, digested at each end in this time are given in the accompanying table. The letters are the same as those in Figures I. and III. (*a*) and (*b*).

TABLE A.

Case.	Samples.						Remarks.
	1	2	3	4	5	6	
A	—	—	—	—	—	1	
B	—	—	—	—	—	3	Trace of free acid.
C	3	4	5				
D	—	—	4	5	6		Trace of free acid.
E	1	—	0				Cancer of stomach.
F	—	—	—	—	—	2	
G	—	—	—	—	—	5	Trace of free acid.
H	0	1	2	3	8		
I	1	1	1	2	3	3	Cancer of stomach.
J	0	1	1	3	3	3	
K	0	0	0	0	0	1	Pernicious anæmia.
L	1	2	1	—	1	1	
M	0	0	0	1	1		Pernicious anæmia.
N	0	0	0	0			Cancer of stomach.

In many there was insufficient material left to work on after other tests had been made. These are indicated in the table by a dash (—).

These then are facts of the case. How can these facts be interpreted? The interpretation must, of

course, depend on the state of knowledge at the present time and on the experience of the investigator and thus these interpretations may be modified with time and in other hands. But the facts remain. It is only by attempting to interpret these facts that we can hope to find the cause of this apparent achlorhydria, a very necessary procedure if we are to treat the condition on correct lines. But in some cases this may be impossible and then we must treat the absence of free hydrochloric acid itself, for as Hurst has recently shown in a most interesting and important paper[2] achlorhydrics are liable to certain special dangers. It is well known that free hydrochloric acid in the stomach acts as a bacteria trap, preventing intestinal infection from food, teeth, tonsils, nasal sinuses and elsewhere. When this free acid is absent, intestinal infection is liable to occur, with production of such diseases as pernicious anæmia, subacute spinal degeneration, some of the chronic joint affections and possibly, as Hurst suggests, appendicitis. That achlorhydria precedes some of these diseases is well known and I cannot do better than refer the reader to Hurst's paper in this connexion.

In treatment Hurst, apparently on the assumption that no hydrochloric acid is secreted by the stomach, gives his patients hydrochloric acid with their food and recommends 5.3 mils (one and a half drachms) of dilute hydrochloric acid in 170 mils (six ounces) of water, flavoured with sugar and lemon-juice, as a drink to be taken with all meals. It seems to me, however, that such treatment can only lead to a further destruction of pancreatic alkali and a continual depletion of the body's alkali reserve. Whether enough alkali would be destroyed to affect health it is impossible to say, but I would hesitate to give this treatment to patients with pancreatic disease, which Bell[3] has found to be often accompanied by achlorhydria. In those inclined to diabetes the overaction of the pancreas might hasten the onset of the disease, if it is true, as F. J. Allen maintains, that pancreatic internal secretion varies inversely as its external secretion.

A much more rational method of treatment would appear to be the prevention of this alkaline reflux by reversing the reflux force during the early part of digestion. Whatever the ultimate cause of the excessive reflux, it is obvious that in these false achlorhydria cases the backward driving force in the duodeno-jejunum must be greater than the emptying force exerted by the stomach, that is the gastric tension. How this comes about we can only guess, but it seems probable that there would be present one or both of the following conditions: (*a*) A low tone of the gastric muscle, allowing fluid readily to re-enter the stomach and (*b*) an irritable small intestine. The first portions of food leaving the stomach would set up a vigorous contraction and peristalsis or raise the tone in the duodeno-jejunum which would thus tend to send part of the food forward more rapidly than usual and part backwards mixed with pancreatic juices into the stomach, with resulting neutralization of its contents.

What evidence have we in support of this supposition ?

In the case of the stomach it is stated that low acidity and achlorhydria are commonly associated with gastric hypotonicity. In my own series of patients there are fifty with achlorhydria of whom twenty-two have been examined with X-rays. Seven of these or 32% have hypotonicity, a figure which approximately equals the percentage of hypotonic stomachs in routine examination. In the last two hundred patients examined by X-rays at the Melbourne Hospital 33% were hypotonic, 58% orthotonic, 5% hypertonic and 4% atonic. It appears then that stomach tone and achlorhydria have very little connexion.

In the case of intestinal irritation it is extremely difficult to disentangle cause and effect, since achlorhydria may allow intestinal infection to occur and conversely intestinal infection and irritation may apparently produce achlorhydria. The only direct case that I have of intestinal irritability producing a back-flow into the stomach is that charted in Figure V. A young woman, *ætatis* twenty-three,

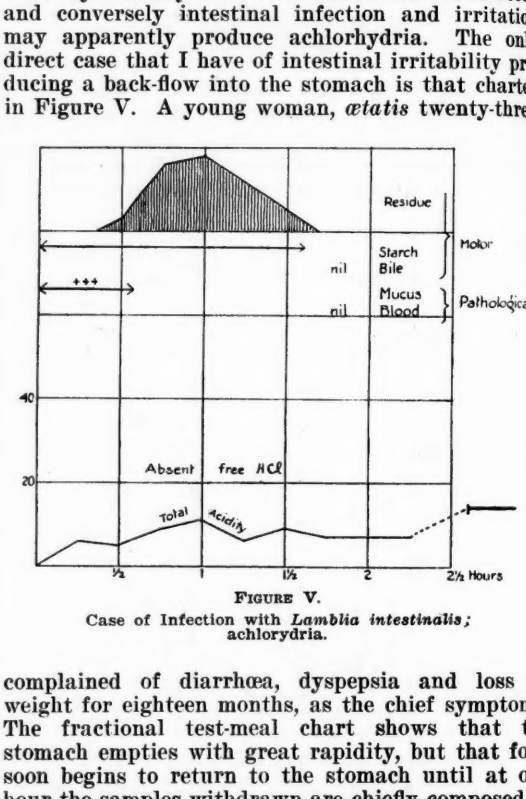

FIGURE V.
Case of Infection with *Lamblia intestinalis*; achlorydria.

complained of diarrhœa, dyspepsia and loss of weight for eighteen months, as the chief symptoms. The fractional test-meal chart shows that the stomach empties with great rapidity, but that food soon begins to return to the stomach until at one hour the samples withdrawn are chiefly composed of food. Then the food residue gradually diminishes again until the stomach is finally empty of all food. This suggested some irritability of the upper part of the small intestine. As a result of this, I had an examination made of the stools and numerous cysts of *Lamblia intestinalis* were found. As this protozoon is known to inhabit the duodenum and upper part of the jejunum, I have some justification for looking upon this as the cause of her reflux and achlorhydria. It was this case that first suggested this investigation into achlorhydria.

On making a closer examination of all these achlorhydria patients it was found that they could be divided into two more or less sharply defined

groups: (*a*) Those in which the stomach emptied rapidly (less than one hour) and (*b*) those in which there was slow emptying (one and a half hours or more). A complete analysis of these groups showed the following as the most outstanding features.

In the rapidly emptying group pain or discomfort often comes on in the first hour after meals (48%) and only a few are relieved by food or drink (8%) or by alkalis (16%). Vomiting occurs in 32% of patients with relief of pain in more than half. Diarrhœa is recorded in only 16%. Of fifteen patients examined by X-rays only 20% had low tone stomachs, the rest being orthotonic.

On the other hand, in the slowly emptying group pain occurs in the first hour in 35%, but in 20% between one and five hours after meals and many are relieved by alkalis (45%) and 20% by food or drink. Vomiting occurs in 50% with relief in two-thirds of the patients, while diarrhœa is recorded in 55%. Of the seven patients examined by X-rays in this group, four had low tone stomachs.

Now it would be expected that those patients who have only apparent achlorhydria, due to complete neutralization by duodenal alkali reflux, would correspond to the slowly emptying group and, if this is so, then the above analysis suggests a cause for this reflux. Thus, it appears that diarrhœa is recorded in the histories of 55% of patients, showing that intestinal irritation was present and was the most probable cause of the more rapid passage of intestinal contents below the point of irritation, as well as the reflux above the point of irritation. Though only seven patients were examined by X-rays, four of them showed a low gastric tone, which would facilitate such a reflux, but, of course, this number is too small to justify any conclusion being drawn.

Another curious point about this group is that in nearly half the patients the pain or discomfort is relieved by alkalis, in spite of the absence of free hydrochloric acid, Recently other writers in America have drawn attention to this alkali relief of patients with achlorhydria and one finds difficulty in accounting for it. The following suggestion may go somewhere near the mark. It is obvious that the pain is not due to acid in the stomach, but relief by alkali strongly suggests that acid is present in some other part of the gastro-intestinal tract. Now Cammidge[4] has pointed out that in patients whose pancreatic juice is absent from the intestine, as in pancreatic disease and I would suggest where a large portion of it is regurgitated into the stomach, abnormal changes occur in the food materials which may interfere with absorption, produce unusual products of digestion which irritate the intestinal wall, and modify the intestinal flora, so that germs which ordinarily exist further down the intestine, may now flourish in the duodenum. Hence organic acids, gas and other irritating products may be formed, producing discomfort and pain two to three hours after food and later diarrhœa. My suggestion is that alkali given after meals in these cases either directly neutralizes these organic acids or, by neutralizing the gastric acid, spares the pancreatic alkali which performs the same function with relief of pain or discomfort.

It might be objected that the diarrhœa in these cases is the result of infection following achlorhydria, but this objection is rendered untenable by the fact that diarrhœa occurs in a very much smaller proportion of patients with achlorhydria in whom there is a rapid gastric evacuation, and also that the patients with slowly emptying stomachs would not have achlorhydria were it not for the intestinal irritation which produces the reflux.

Referring again to Hurst's paper[2] it is interesting to note that he regards many cases of appendicitis as being due to intestinal infection following achlorhydria. I am inclined to believe that many of these achlorhydrias are also due to the intestinal irritability accompanying an inflamed appendix. Bonar[3] found in sixty-five patients with appendicitis that 55% had hyperchlorhydria and 33% had achlorhydria. The former were due, of course, to pyloric closure or hypertonus, produced by distant reflex action, while it is probable that the latter were due to the more local bowel irritability. As we advance higher in the intestine, where local irritability is closer to the stomach and would therefore be more likely to produce an alkali reflux into that organ, we find that the proportion of accompanying achlorhydria is higher, as we would expect. Thus the same writer found that gall-bladder disease is accompanied by hyperchlorhydria in only 23% of patients, but achlorhydria has now risen to 49%.

I am at present treating certain of these patients with false achlorhydria on the lines indicated, namely (i.) by attempting to remove the sources of intestinal irritation and to allay intestinal irritability and, (ii.) by raising gastric tone rather than by Hurst's method of giving hydrochloric acid, a method with which I have not been greatly successful. The bitter tonics which rouse appetite and therefore raise gastric tone, are, of course, familiar in the treatment of hypochlorhydria, a condition commonly due to increased intestinal reflux. These results will, I hope, be published at a later date.

Conclusions.

(1) The majority of patients with apparent achlorhydria have a normal secretion of gastric acid, but this is neutralized as rapidly as it is secreted, owing to an abnormally large regurgitation of duodenal alkali.

(2) While it is as yet impossible to state the exact cause of this increased reflux, there is evidence that it is due to increased intestinal irritability.

(3) Although the evidence suggests that absence of free hydrochloric acid in these cases is merely one of the signs of an intestinal indigestion, it is important to treat this sign, since loss of free gastric acidity may lead to intestinal infection with serious consequences.

(4) Treatment in these cases should be directed against the cause of the intestinal irritation and should aim at the prevention of duodenal reflux by allaying this irritability and raising gastric tone.

(5) In cases of *carcinoma gastrica*, true gastritis and some forms of pernicious anæmia the experimental results show a definitely decreased secretion of acid, but a surprisingly large number of patients with pernicious anæmia have a good secretion of acid.

Acknowledgments.

My thanks are due to Dr. S. W. Patterson for permission to work in the laboratories under his charge, to Dr. Gordon Cameron for his valuable help in the tedious work of chloride estimation and to Dr. R. H. Strong for his help and advice in the treatment of these cases.

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OCULAR EMERGENCIES IN GENERAL PRACTICE.

BY J. CAMPBELL DOUGLAS, D.O. (Oxon.),
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THE general practitioner and particularly the lodge practitioner has on many occasions to act the part of ophthalmic surgeon, for if his patients do not expect him to test them for glasses, they generally look to him to save them the trouble and expense of visits to the eye specialist in the minor emergencies of ophthalmic surgery.

And it is in the urgent surgery of eye cases that much comfort to the patient and much credit to the lodge doctor can quickly be obtained, to say nothing of the difference between an eye saved and an eye lost, so far as vision is concerned.

Foreign Bodies in the Eye.

Among the most common and most distressing of minor ophthalmic emergencies are foreign bodies in the eye. These may be situated under the lids, in the upper or lower fornix of the conjunctival sac or adherent to or embedded in the cornea. A small foreign body in the upper *sulcus subtarsalis* will cause more pain and redness than one sticking in the cornea itself, for every movement of the lid in winking will drag it across the sensitive cornea.

The first place to look for a foreign body is beneath the upper eyelid. Here it will generally be found beneath the centre of the lid in the groove which runs from side to side just behind the lid margin. If not found in this situation, the upper fornix must be exposed by double eversion of the eyelid. This is accomplished in the following way. While holding the lid everted by the thumb on the eyelashes, gently press on the eyeball with the fingers of the other hand through the lower eyelid in a backward direction. The loose folds of the upper fornix will start forward suddenly and can be searched and examined. Quite large foreign bodies can remain concealed here for days and weeks.

I can recall an amusing incident which happened while I was studying at a London eye hospital. One morning the professor arrived early before any of the students and saw a man who had come complaining of a sore eye. After examining him carefully he put him aside to wait. Later in the morning when he had a dozen students around him, he called the patient up and choosing the brightest student from the group, one who rather fancied himself as a diagnostician, invited him to examine the case and tell the class what was wrong with the man. The student spent fully five minutes carefully examining the eye without finding anything beyond the small amount of redness and injection. He even everted the upper lid once without gaining any fresh knowledge, but he did not doubly evert it. He suggested in a diffident way that the patient had had something in his eye which had been washed out with tears. Then the professor with a sly wink at the other students everted the lid, then everted it a second time, when there started forth from the upper recesses of the fornix a large tasselled grain of wheat, over a centimetre long. Even the unfortunate student was forced to join in the roars of laughter which greeted his discomfiture.

Foreign bodies under the lids are easily removed with a clean handkerchief or an eye spud and no cocaine is necessary.

It is different on the cornea. Here the foreign body is usually very minute and there may be difficulty in locating it. Place the patient in a chair near the window, for a good light is absolutely essential. Examine the eye first from one side of the patient, then from the other, using a bi-convex lens to focus the light on the cornea and a pair of reading glasses if you are presbyopic or a binocular *loupe* for magnification. If it is not easily found a drop of solution of fluorescein applied on a glass rod to the upper part of the eyeball may show the slight injury to the corneal epithelium by staining it a bright green colour. The spot of green can then be closely examined with the magnifying glass.

When the foreign body has been located, a few drops of 2% cocaine solution should be instilled. Then standing behind the patient, the surgeon holds the lid apart with the finger and thumb of the left hand, while with the right he removes the foreign body, aiding his vision with the binocular *loupe*. And here I can offer a useful hint. First of all try to remove the small speck with the aid of a piece of filter paper shaped somewhat like a broad J nib. Quite a number of foreign bodies are easily removed in this way with minimum damage to the corneal epithelium. If this fails, try a blunt spud, gently prizing the speck off the cornea. Only if this second method fails should you take up the flat pointed discission needle and lever out or scrape out the offending particle according to its nature.

In the case of a particle of steel do not fail to remove the rust staining which often remains when the foreign body has come away. If you fail to do so, the patient may not get complete relief; he may return for a second operation or go elsewhere with a dissatisfied mind. The whole operation from start to

finish should be done with the greatest delicacy and gentleness. Remove no more of the corneal epithelium than you can help; do not increase the damage already done by the foreign body.

Finally wash out the eye with a little warm boracic lotion, instil one drop of homatropine (not atropine) to dilate the pupil slightly and keep the ciliary body at rest and apply a bandage for twenty-four hours.

This is necessary after using cocaine.

The eye should be inspected on the following day for fear of infection, but if the eye is then quiet and looks well, the patient may be told to bathe it himself for a few days with boracic lotion.

Corneal Ulcers.

The condition produced by a foreign body in the cornea might be termed an aseptic traumatic corneal ulcer. If all goes well it should heal without inflammatory reaction, but if it becomes infected then an infected or septic corneal ulcer developes. Sometimes corneal ulcers form without traumatism, as in scrofulous disease and after injury to the fifth nerve and in people whose health is below par. However it may occur, an ulcer of the cornea is always a source of grave danger to the eye, but if promptly and correctly treated by the general practitioner it very soon gets well. A favourable response to treatment can often be seen within a few hours. If improperly treated or neglected, hypopyon or pus in the anterior chamber frequently forms and then the eye is in a very dangerous state indeed and the patient should be referred to a specialist.

Many different types of corneal ulcer are described in the larger text-books on ophthalmology, but the differential diagnosis need not concern the general practitioner; the broad general principles of treatment are the same in all.

These fundamental principles may be summed up as cleanliness, rest, heat and protection. Cleanliness is attained by the use of antiseptic lotions, heat by means of foments, rest to the internal parts by the use of atropine and protection by a pad and bandage lightly applied. Four great enemies of a damaged cornea are wind, dust, smoke and glare and the bandage protects against all of these.

The details of treatment are as follows. The conjunctival sac should be washed out three times a day with warm boracic lotion (1.8% or eight grains to the ounce). The use of stronger antiseptics than boracic are of little benefit. An "Undine" which is a flask-shaped glass bottle, is the best means of applying the lotion, but dripping it in from a basin by means of a pad of wool or pouring it in from a small china teapot or an invalid feeding cup would do equally well.

After the irrigation one drop of a 1% solution of atropine should be instilled into the eye or an ophthalmic disc of atropine placed inside the lower lid. Atropine does good in two ways. It keeps the eye at rest by paralysing the ciliary muscle and rest is a great means of relieving pain, as Hilton showed us long ago. In the second place it dilates the pupil and so prevents iritis and the adhesion of the iris to the under surface of the corneal ulcer. Adhesion between the iris and the cornea is a very dangerous complication. As always when using atropine watch the tension carefully.

Next a pad and bandage should be applied. Special oval pads to fit the eye can be obtained, but cotton wool or Gamgee tissue is equally good. A single turn of bandage is sufficient. The bandage should be 7.6 centimetres broad. It is not necessary to roll it around the head. It should be neatly arranged obliquely over the pad and head and tied beneath the occipital region. No pressure should be used as pressure sometimes causes an ulcer to rupture into the eye. If the ulcer looks septic and the eye red, then it should be sterilized with pure carbolic acid. I believe that 95% of all corneal ulcers can be carbolized with only good results. The application should not be made more than once, as a rule.

The method of application is as follows.

Cocaine solution (2%) is instilled twice or three times. The doctor stands behind the patient's chair and separates the lids with the fingers of one hand. The surface of the ulcer is dried with a strip of filter paper. A wooden toothpick is soaked in the carbolic acid until thoroughly wet, but not even the slightest bead or drop of the acid is to be hanging from it. This is then gently applied to the surface of the ulcer and lightly stubbed into the edges all round. It is in these edges that most of the organisms are.

If there is difficulty in seeing the extent of the ulcer, one drop of fluorescein may be instilled after the cocaine; this makes the ulcer stand out bright green.

Finally the ulcer is again touched with the filter paper to dry off any surplus carbolic acid. Even if a tiny drop of acid should spread on to the surrounding corneal epithelium, it would not do any harm beyond leaving a temporary white mark.

Hot foments should be applied by the patient at home or hot bathings with boracic lotion at intervals of four hours. This eases pain, lessens stasis and assists repair. A useful tip for applying heat is as follows. Direct the patient to sit down with a basin of fairly hot water or boracic lotion. A lump of cotton wool is placed inside a tablespoon and the hot water is lapped up with this and held against the eye for half a minute, then another spoonful. The application should be continued for a quarter of an hour and the pad and bandage applied. Or dry heat may be continuously applied while the patient lies in bed by means of an electric light bulb on the end of a long piece of flexible. First apply cotton wool to the eye, then over this place the bulb with the light turned on, then a single turn of bandage lightly applied. Quite a lot of heat is obtained in this way.

Drops of "Collosol argentum" or drops of "Protargol" (15%) may also be given to the patient to use at home. They should be used immediately after the irrigation with boracic. For the scar which is

always left after an ulcer has healed, the long continued use of 1% *unguentum hydrargyrum oxidi flavi* gently massaged into the scar through the closed lids is of great benefit. A year or eighteen months is not too long to use it. The patient should be taught its use. I have seen a patient squeeze out enough for half a dozen eyes at a time. Only the tiniest fragment should be squeezed out and flicked off with a bone crochet needle or smooth glass rod before applying. It reminds me of the mustard manufacturer who said he got rich from the mustard that people left on their plates, not from the small amount they consumed.

More drastic methods of dealing with corneal ulcers and their complications should be left to the eye specialist to apply. Corneal ulcers which are not going right, which are progressing in the wrong direction, contain so many possibilities of grave disaster to sight that the practitioner for his own peace of mind is well advised to refer them to an ophthalmologist.

Bandaging an Eye.

It is a matter of doubt sometimes to the general practitioner when to bandage an eye and when not. Bandages are used in eye cases for three purposes: to exclude light when there is photophobia, to shut off a wound from contact with the outer air and in a few cases to exert pressure upon the globe.

Generally speaking a bandage should not be applied for conjunctivitis or conjunctival affections, but should be applied for corneal or deeper affections such as iritis. An aphorism used by a London professor was "In conjunctival injections don't bandage, in ciliary injection do"

Even in corneal ulcers, if there is much purulent conjunctival discharge associated with it, a bandage does more harm than good. A shade is better. This can be made in a few minutes from a piece of brown paper of the size and shape of the outline of a kidney tray (the latter may be used to mark it out in pencil), with a strip of the same paper to go round the head and caught through a slit at each side of the shade.

The celluloid single eye shade sold in the shops is too hot and stuffy, is not as valuable as a proper bandage in ulcers and is detrimental in conjunctivitis. Seven and a half centimetres is the proper width for a single turn bandage, six and a quarter centimetres for a roller bandage, five centimetres in a child. A useful tip is to dip it in hot water and ring it out while still in the roll. It will then sit more comfortably and firmly, if firmness be desired or in children after operation. In simple dressings always remember to let the bandage be lightly applied.

Drops.

When instilling drops into an eye tell the patient to look up, pull down the lower lid and let the drop fall into the lower fornix and not on to the sensitive cornea which would startle the patient and make him flinch. The dropper should not be held too near the eye for two reasons. You do not want it to brush against the lids or eye lashes for it is no longer aseptic and, if it is held too close to the cornea, it might easily do serious injury to the eye if the patient suddenly raised his head. About five centimetres is a good distance.

Flask-shaped glass bottles with glass cups are best; they hold sufficient fluid and if held in the heat of the hand, the drop comes out more easily. A simple form costing one penny each is obtainable in England, but not in Australia, though I have seen a good Japanese drop bottle at twopence.

All eye drops can be sterilized by boiling in a small test tube. The drops in common use are atropine, homatropine, eserine and cocaine. Atropine drops are most powerful and dangerous of all drops. They are used in a strength of 1%; their effects are not produced for several hours and do not pass off for fully a week. They are used in corneal ulcers, in iritis and in chorioiditis and in testing children for spectacles. They should not be used in conjunctivitis as is often done. Indeed atropine is too often used and in unsuitable cases by some practitioners.

The tension of the eye should always be tested with the fingers before drops are instilled. Taking the tension of an eye is not always properly done. The forefinger of one hand should steady the eyeball and hold it in position without moving, while the forefinger of the other hand seeks to dimple the eyeball from above. Both sides should be compared.

Homatropine is much safer than atropine, for its effects can be overcome with eserine. It is commonly combined with cocaine. The prescription is homatropine 1%, cocaine 2% in solution. It is the best drug to use for patients between fifteen years and forty-five years of age.

"Euphthalmin" 2% or scopolamine ¼% are better drugs to use in older people, if one merely wishes to examine the *fundus oculi* or after removal of small foreign bodies. They are safer at an age when glaucoma is a possibility to be considered.

Eserine in ½% solution is employed to contract the pupil. If used too often or too strong, it causes pain in the eye and headache. One drop only should be instilled till the effect is noted. It overcomes the effect of homatropine and "Euphthalmin," but has no effect upon atropine dilatation. It is used chiefly in glaucoma. Cocaine and its substitutes, "Holocaine," "Novocain" and "Eucaine" are used to anæsthetize the cornea and conjunctiva before operations, 2% and 4% solutions being commonly used. When added to solution of homatropine it helps diffusion of the latter drug through the cornea. After instilling cocaine solution for small operations on the lid such as that for chalazion, a little solid "Novocain" should be rubbed into the site of the incision with a spud. This makes the operation nearly painless. The incision is then made with a Beer's knife, a little solid "Novocain" put inside the cyst with the tiny Volkmann spoon which is used for scraping out the cyst wall. This latter must be done in all these cases or the chalazion will return.

After using cocaine an eye should be bandaged for a few hours, for the patient has a much lessened tendency to wink on account of the anæsthesia of the cornea and the latter tends to become dry and its epithelium to exfoliate. This especially applies after the removal of a foreign body under cocaine.

Glaucoma.

Four acute conditions may be met with in the ophthalmic work of a general practice. They are injuries to the eye, corneal ulcer, acute iritis and acute glaucoma. Of these the last named is the most acute and the most urgent. All the other conditions may be left for many hours unattended and, if then properly treated, the result will probably be good. Acute glaucoma cannot wait. In a few hours sight is destroyed for ever. It is the one condition for which the ophthalmic surgeon is called to the hospital in the middle of the night. Every general practitioner should be able to recognize and to suspect the condition when seen. The diagnosis is not always easy. I have known it mistaken for "acute abdomen" and the abdomen nearly opened. I heard a lecture by a London surgeon on the differential diagnosis between acute glaucoma and acute appendicitis. In both there is vomiting, a raised temperature, a running thready pulse with signs of collapse, the patient is in bed and much too ill to answer questions or to permit a leisurely examination. The eyes may be kept closed and the loss of sight not noticed by the patient. The redness of the eye may be put down to the strain of vomiting, the dilated pupil to the darkened state of the room.

It has also to be distinguished most carefully from acute iritis in which the symptoms are somewhat alike, but for which the treatment is exactly opposite.

Once on a London station I saw an elderly lady collapse on the platform from the sudden onset of an acute glaucoma. She was quite unable to move and had to be taken by ambulance to the hospital for operation.

When examination is permitted, the injection of the eyeball, the great pain, the tenderness on touching, the dilated, immobile pupil, the greenish haze over the latter and the hardness of the tension will point the way to the true diagnosis.

Eserine drops (1%) should be instilled as early as possible. If this does not relieve the tension somewhat, then a straight surgical needle should be sterilized and thrust carefully into the vitreous chamber of the eyeball just behind the equator. A whiff of chloroform is necessary for this. The needle is twisted round and slowly withdrawn to let some fluid escape. Then the eserine (½% to 1%) and hot bathings should be continued until the patient can be subjected to operation.

Loss of Vision.

A patient may present himself to the doctor with considerable defect of vision in one or both eyes. Is it due to an error of refraction or to some serious pathological process? A simple test is with the pinhole disc. This can be made by the doctor in a moment by pushing a darning needle through a visiting card. If the patient by using this in front of the eye which is being tested, can read one or two lines more of the test type, it is most likely a simple case of refraction. If vision is not improved, it is probably a serious disease of the retina, or chorioid or optic nerve. And whether it is the one or the other, the lodge practitioner with a busy practice and working against time will be well advised to turn the case over to his specialist colleague.

Every such case without exception demands a complete examination of the eye in all its parts; the pupil must be dilated, the refraction carefully estimated, the different media and the *fundus oculi* thoroughly explored with the ophthalmoscope and possibly in addition the fields of vision mapped out with the perimeter, as in retro-bulbar neuritis and pituitary disease.

All this takes time which the average doctor is ill able to spare and demands for the correct interpretation of the signs the experience of a large number of cases. Even the estimation of the refraction with any degree of accuracy requires constant practice and the judgement born of experience, as the large number of partial failures in the hands of even experienced opticians and ophthalmic surgeons bears witness.

While the correct prescription of spheres in a case of simple presbyopia is a matter requiring no great skill or special knowledge, yet in the majority of cases of errors of refraction many other things have to be taken into consideration and allowed for. These include such things as the testing of the motor muscle balance of the eyes, the presence of esophoria or exophoria, the various tests for latent deviation, the placing of the *punctum proximum* at the correct distance for near vision, the suitability of various spheres for different occupations and the judgement by retinoscopy of the exact moment of the reversal of the shadow and the exact axis of astigmatism. These things mean all the difference between comfort and discomfort to the patient. In short, the gain is not worth the risk.

If, however, in a suspected case of ocular headache the practitioner wishes to be able to tell his patient whether he has myopia, hyper-metropia or astigmatism, then a very simple outfit and a few minutes only are required. Obtain a pinhole disc, a stenopaic slit, a one diopter convex lens, a — 2.0 diopter concave lens, a distance test type, a near-vision Jaeger reading type and a clock face card or set of astigmatic lines. All these can be obtained for a few shillings.

Place the patient at six metres (about twenty feet) from the distance test types which must be hung on the wall in a good light. See how many lines he can read with each eye separately. If he misses some of the lower lines, try him with the pinhole disc. If more lines can be read with this, he has an error of refraction. Then try the + 1.0 convex sphere. If this improves vision and makes him read another line, then he has hypermetropia. If vision is made worse by the + lens, try the

— 2.0 diopter concave lens. If this enables an extra line or two to be read, then there is probably myopia. Now try the stenopaic slit, with the clock face card on the wall. If some of the figures on the clock are less distinct than others, the stenopaic slit turned in certain directions will make these figures more distinct. This proves the presence of astigmatism which may be combined with either myopia or hypermetropia. If the patient is over forty years of age, try him reading the near vision card at thirty centimetres (twelve inches). If he cannot read No. 1 or No. 2 Jaeger type at this distance, put up the + 1.0 convex lens and, if with this assistance he can now read No. 1, then he has presbyopia or loss of accommodation. These are useful rough tests which can be easily and quickly applied.

Squint.

When a child developes a squint, the mother usually consults the family doctor in the first place. Sometimes he is at a loss to know the best advice to give in these cases. The matter, however, is a simple one. If the squint has only existed for a few months, that is if it is an early case, then the refraction should be estimated and glasses prescribed. Squint has a natural tendency to disappear as a child grows older. This is greatly helped by careful and full correction of any refractive error and this latter treatment alone will often cure a squint.

Treatment by glasses should always be tried for six to twelve months and re-examination made to see if there is any improvement. If there is decided improvement, continue the treatment and assist it by exercises with the amblyoscope or steroscope to develope the fusion sense. If there is no improvement, then it is wiser to operate before it is too late and the eye amblyopic (permanent weak sight).

Six years is the critical age. If the squint has not been put right or nearly so before then, the macular region of acute vision and the power to fuse the two images in binocular vision will show little improvement or development after this age.

Glasses may be worn at any age from six months onwards for squint. I have had babies of nine months wearing them with great satisfaction and without any attempt at pulling them off.

After six years of age the need for operation is less urgent, the mischief is already done. Glasses can then be worn until the age of ten years and the operation performed under cocaine. The correcting spectacles are worn after every operation for squint and should be ready in advance.

Some surgeons, but not the majority, believe in operating about two years of age in every case of squint which has shown itself by then. The operation sets the eye straight, the glasses then cure the cause of the squint.

Various operations are carried out for squint. The tendon may be cut across (tenotomy). Cuts may be made in the sides of the tendon (tendon-lengthening operation). The tendon may be divided and brought forward to a new attachment (advancement operation). The muscles may be reefed or tucked (reefing operation). If a squint is left for Nature to cure or if in a child seen before six years operation is postponed until nine or ten years, then permanent damage to the squinting eye results (*amblyopia ex anopsia*). Nothing can be done for this in later life.

If you find an adult with one eye normal and the other eye very defective, you will often find that the defective eye squinted in early youth.

Reports of Cases.

CASE OF TUBERCULOUS MENINGITIS WITH EXTREME NEUTROPHILE LEUCOCYTOSIS.

By HARRY J. CLAYTON, M.B., Ch.M.,
Assistant Honorary Physician, Royal Prince Alfred Hospital.

Pathological Notes.

By MONA ROSS, M.B., Ch.M.,
Chief Resident Pathologist, Royal Prince Alfred Hospital.

THE following case is of special interest because of the extreme neutrophile leucocytosis somewhat obscuring an otherwise complete picture of tuberculous meningitis.

T.T., aged thirty-one years, furniture maker, was seen on June 12, 1923, in consultation with Dr. Kevin Byrne. The patient had lead an exceedingly strenuous life, acting outside business hours as "sparring partner" and teacher of boxing. There was no family history of any importance and none of the members had suffered from tuberculosis.

The patient had suffered from enteric fever five years previously, but had been otherwise quite healthy. There was no history of venereal disease.

In February he had consulted Dr. Byrne, complaining of general ill-health, no appetite, loss of energy *et cetera*, but the symptoms were very general and no clinical evidence of disease was found. He did not report again, but his wife stated that he did not improve and had been complaining ever since of general disturbance of his health. For nine weeks prior to June 9, 1923, he complained of continuous headache in the occipital region and of sleeplessness, which symptoms became very severe and prostrating in the previous four days. On June 9, he became delirious and was seen again by Dr. Byrne. His condition became gradually worse and on examination on June 12, his temperature was 37.3° C. (99.2° F.), his pulse-rate 62 and his respirations 22; he was semi-comatose and unable to respond to questions. His clinical picture was typically that of the "typhoid state," but in addition he was extremely irritable, lying curled up in bed and resentful of interference and examination.

Under these circumstances it was difficult to conduct a thorough examination, but no definite abnormal clinical signs were found in the abdomen. There was retraction at the apices of both lungs, more particularly the right, but percussion and auscultation revealed no obvious change. The arteries were much thickened and there were signs of hypertrophy and dilatation of the heart, but with no evident valvular or aortic lesion.

Signs found in the nervous system were his "typhoid state" and extreme irritability. Sensation and coordination could not be tested. The cranial nerves were apparently intact. There was no loss of power. No reflexes could be elicited owing to the patient's irritable condition increasing the difficulty of examination. There was no head retraction, but there was definite hypertonus of the neck (not amounting to rigidity) and there was evidence of pain on bending the head forward. Hypertonus was also present in the lower limbs together with pain on flexion of the thigh on the abdomen, though here again the amount was not so great as to constitute an obvious Kernig's sign.

A differential diagnosis of (*a*) meningitis, probably tuberculous, possibly syphilitic, (*b*) encephalitis with meningitis, (*c*) cerebral tumour with supervening hæmorrhage was made with preference in the order given.

The patient was admitted to hospital immediately and the following further examinations were performed. On lumbar puncture forty cubic centimetres of clear fluid under increased pressure were withdrawn under anæsthetic. There was no chemical alteration and no increase of cells; no organisms were present. The Wassermann test did not yield a reaction. No reaction to the Wassermann test with the patient's blood serum was obtained. The leucocyte count was as follows:

Leucocytes, 21,000 per cubic millimetre.
Differential Count—

Neutrophile cells	80%
Lymphocytes	15%
Large mononuclear and transitional cells	5%

The red cells were not counted.

After lumbar puncture the patient showed marked improvement and his temperature, which on admission to hospital was 38° C. (100.4° F.), gradually came down and the pulse-rate increased, with improvement in mental condition and loss of hypertonus.

Sixty hours after admission he was to all appearance normal mentally and said he felt quite well. The pulse and temperature were normal and he was asking when he could leave hospital.

Twenty-four later, however, the symptoms gradually recurred and five days after his admission he was in the same condition again. A lumbar puncture then yielded seventy cubic centimetres of cerebro-spinal fluid under increased pressure with the same characteristics as previously. Repeated twenty-four hours later, ninety cubic centimetres were withdrawn, with no additional pathological findings of diagnostic value.

Twenty-four hours later he died.

Pathological Report.

At autopsy in the respiratory system there were dense adhesions over the whole area of the right pleura, more evident at the right apex. There were adhesions also at the left apex. Both lungs were in a condition of acute congestion, the right being the more affected. On palpation areas of consolidation were found in the right lung. On section of the lungs a cavity, the size of a large walnut, was found at the right apex, filled with caseous material and one about half the size at the left apex also. There were scattered miliary nodules throughout the right lung and at the apex of the left lung.

In the left ventricle there was a considerable degree of hypertrophy. No other abnormality was present in the cardio-vascular system. The spleen was enlarged and congested and the liver was congested. Both kidneys were subacutely congested; the capsules stripped freely. The right kidney contained several scattered miliary nodules and there was one nodule in the left kidney on section.

On removing the brain from the cranial cavity a large quantity of turbid cerebro-spinal fluid escaped. There was marked engorgement of the cortical vessels, particularly in the occipital regions. At the base of the brain the meninges were adherent and matted together by purulent exudate. The Sylvian fissures were filled with a similar exudate and on separating the opposing surfaces, the meninges were found studded with multiple miliary nodules along the course of the vessels.

The spinal cord was not examined.

In the smears taken from the exudate there were tubercle bacilli in great numbers. These were also present in smears taken from the nodules in the lungs.

Seventy-two hours before death the temperature reached 39.4° C. (103° F.) and remained elevated, while the respirations increased to thirty-two and later thirty-six. Evidence of pneumonia or miliary tubercles was then sought for in the lungs, but none was found, though there could be no doubt that a pneumonic process had complicated the original picture.

The old lesions found at the autopsy at the apices were not at any time detected clinically, except in the retraction, more evident on the right side, though several examinations were made, but these were perhaps not satisfactory as it was difficult to examine the apices by percussion and auscultation in the irritable condition of the patient.

The presence of such a lesion as a primary focus, in view of the provisional diagnosis was, however, suspected and examined for as carefully as was possible under the circumstances.

The extraordinary feature of this case which makes it worthy of record, is the extremely high neutrophile leucocytosis, tending to obscure an otherwise excellent picture of tuberculous meningitis.

A neutrophile leucocytosis of 10,000 to 12,00 is quite common in acute miliary tuberculosis (and tuberculous meningitis) and is indeed to be expected. Türk reports a case with 20,8000 leucocytes as the highest found by him and this is the only record I can find approaching the case now reported. There are numerous reports of between 10,000 and 14,000 and a leucocytosis, neutrophile in type, is reported in approximately half the cases examined.

The explanation in this case may be, of course, that the caseous pneumonia present at autopsy was developing (with perhaps a mixed infection superadded) at the time the blood count was taken, but at that time there was not only no evidence of this clinically in the lungs but, in addition, the temperature was coming down (it had not been higher than 38° C. *id est* 100.4° F.), the pulse rate was not above normal, the respiration was not increased and there was no cough or sputum. In fact there was no evidence whatever of acute involvement of the lung by the tubercle bacillus or any other organism.

Dr. Mona Ross, who carried out the pathological examinations and the autopsy, has been good enough to contribute the description of the morbid anatomy in this case and to her and to Dr. Byrne I wish to extend thanks for the interest derived.

Reviews.

MINOR SURGERY INCLUDING BANDAGES.

"MINOR SURGERY INCLUDING BANDAGING," by Dr. Henry R. Wharton is an excellent little book.[1] The work is divided into seven parts. This makes the book more readable and facilitates reference.

In the first part the author gives a good description of bandaging. This is fast becoming a lost art. The many illustrations will be helpful to students. It is a pity that the author has not included a description of the celluloid splint.

The second part on minor surgery is well written and comprehensive. A great deal of ground is covered and much information is given. There are some points, however, to which exception may be taken. For example, the author states on page 188 that electrolysis has been applied with success in the treatment of hydatids. The statement on page 213 that "Stovaine" hydrochloride is dangerous as an anæsthetic is open to question, while many surgeons of experience will hesitate to accept the author's advice in regard to the injection of cocaine into the urethra. Likewise the advice in regard to the incision and drainage by means of a tube of tuberculous abscesses requires to be qualified. The same illustration has been used to depict the reef and granny knot.

In the third part the author covers the field of asepsis and antisepsis and gives a short account of surgical bacteriology. All the modern drugs which came to the fore so much during the late war, are described and their merits discussed.

[1] "Minor Surgery, Including Bandaging," by Henry R. Wharton, M.D.; Ninth Edition, thoroughly revised; 1923. London: Baillière, Tindall & Cox; Demy 8vo., pp. xii. + 647, with 450 figures in the text. Price: 18s. net.

The fourth and fifth parts are devoted to fractures and dislocations. So much has been written on these subjects in recent years that the author is to be congratulated on so apt a presentation of the subject in so small a space.

Parts six and seven on minor operations and the ligature of arteries are written on conventional and well established lines.

In spite of the few inaccuracies to which attention has been drawn, there is so much that is good in this little book that we recommend it with confidence to students and practitioners.

LABORATORY STUDIES IN TROPICAL MEDICINE.

THE fifth edition of Daniels and Newham's "Laboratory Studies in Tropical Medicine" is a volume that will commend itself to medical officers throughout the tropics.[1] As the authors very truly remark, there are few institutions in the tropics which correspond to the British idea of a laboratory, and the isolated worker has to manufacture his laboratory in a corner of his office or hospital frequently without the facilities of tap water, gas or electric light. In these circumstances the exhaustive and detailed information contained in this edition will be of considerable value.

The modification of Mann's stain for Negri bodies which is outlined, gives results which are an advance on previous technique. The sections devoted to entomology and helminthology are excellent of their kind.

From an Australian standpoint even more attention could perhaps have been devoted to the difficulties that arise in the use of Leishman's stain, while the formula for nutrient agar is inapplicable in northern latitudes, at least 4% of the powdered agar is required for optimum results.

It would perhaps have been better in connexion with the Wassermann test to have outlined one of the later recognied methods than to have emphasized a method not in common use. The Wassermann test will be done only in central laboratories provided with every facility and not by the local practitioner. Consequently the emphasis might better have been placed on the methods of collection and despatch of the serum.

The outstanding feature of the book, however, is its general practicability and the new edition is an improvement upon the excellence of its predecessors.

POST-GRADUATE LECTURES.

"TEN POST-GRADUATE LECTURES" is the work of eight contributors and has a most illuminating preface by Sir Clifford Allbutt.[2] In book form these lectures possess something of the flavour of an informal armchair chat with the consultant. Too often such publications fail to interest those for whom they are written; on the one hand they repell the general practitioner by their rudimentariness or on the other they overwhelm him by their scientific aloofness from the pressing problems of every-day practice. It is therefore vastly refreshing to meet with a book which is neither a textbook nor a treatise, but just that golden mean which by its matter attracts and by its manner holds the attention.

In the two lectures by him the late George Savage says much that is helpful in the elucidation of the baffling difficulties presented by mental disorders and syphilis and the inter-relation of these diseases. The reader of these two lectures will be fully persuaded of the truth of Sir Clifford Allbutt's estimate of their author as "one of the happiest, wittiest, kindliest and, in his own subject, most experienced members of our profession."

Sir William Hale-White contributes a beautifully expressed review of the prognosis in exophthalmic goitre, the reasoned optimism of which should encourage practitioners to look a little less gravely at this disease and to enhance their belief in the value of rest.

Grave familial jaundice in the new-born receives a very complete and masterly account at the hands of Sir Humphry Rolleston.

The essay by Dr. Hernaman-Johnson on combined treatment by surgery, electricity and X-rays should bring help to the surgeon by indicating methods of avoiding such surgical bugbears as the keloid cicatrix, adhesions in joints near fractures and in the abdomen after section. It should help the physician by suggesting means of stimulating the lagging growth of lower limbs paralysed by polio-myelitis.

The responsibility for the after effects of anæsthesia as between the anæsthetist and the surgeon comes in for appraisement at the hands of Dr. J. D. Mortimer with rather disconcerting results to the latter.

The two longest lectures in the book are by Dr. Trethowan on disabilities of the feet caused firstly by mechanical causes and secondly by paralysis. Both are very accurate and thoughtful expositions of the causation and methods of treating these most difficult and disappointing problems of practice.

A short but opportune lecture on *pyorrhœa alveolaris* and its relationships with disease serves to clarify the somewhat nebulous beliefs held by many on this subject.

In the hands of Dr. Wilfred Harris, the injection of alcohol into the Gasserian ganglion seems a safe and satisfactory method of treatment for chronic paroxysmal trigeminal neuralgia.

The book is in every way excellently produced and is provided with a surprisingly complete index. There are very few typographical errors in the text: the only glaring one noted occurs on page 150, line seven, where "incoordination" obviously should read coordination.

ENLARGEMENT OF THE PROSTATE.

DR. JOHN B. DEAVER has published the second edition of his monograph entitled "Enlargement of the Prostate."[1] Although this book contains much that is old and little that is new, the author lays stress in an admirable manner on the necessity of early diagnosis of enlargement of the prostate and adequate pre-operative treatment if the best results are to be obtained. His collected statistics prove conclusively the benign nature of the operation for removal of the prostate in the hands of urological experts. The mortality in these circumstances is from 3% to 5%. He contrasts with this the fact that in the hands of the average hospital surgeon the mortality is from 10% to 35%.

In regard to cystoscopic examination the author has performed a *volte face*. In the first edition he claimed that cystoscopy was rarely if ever necessary, while in the present edition he states that "the diagnosis of prostatic hypertrophy cannot be said to be complete without a cystoscopic examination." The usual teaching is that cystoscopy should only be carried out when necessary for diagnosis.

The method of palliative cystotomy described by the author, namely that by oblique supra-pubic fistula, is certainly not the best practice.

Cancer of the prostate receives but scant mention. Its treatment is entirely omitted. The operative treatment of this disease has in selected cases yielded most promising

[1] "Laboratory Studies in Tropical Medicine," by C. W. Daniels, M.B. (Camb.), F.R.C.P. (Lond.), and H. B. Newham, C.M.G., M.D. (Durh.), M.R.C.P. (Lond.), D.P.H. (Camb.), D.T.M. & H. (Camb.); Fifth Edition, Thoroughly Revised; 1923. London: John Bale, Sons & Danielsson, Limited; Demy 8vo., pp. xiii. + 550, with index, seven coloured plates and 184 illustrations. Price: 25s. net.

[2] "Ten Post-Graduate Lectures Delivered Before the Fellowship of Medicine at the House of the Royal Society of Medicine, 1919-1920," with a Preface by the Right Honourable Sir Clifford Allbutt, P.C., K.C.B., M.D., F.R.S.; 1922. London: John Bale, Sons & Danielsson, Limited; Demy 8vo., pp. 216. Price: 10s. 6d. net.

[1] "Enlargement of the Prostate," by John B. Deaver, M.D., LL.D., Sc.D., F.A.C.S., assisted by Leon Herman, B.S., M.D.; Second Edition; 1923. Philadelphia: P. Blakiston's Son and Company; Royal 8vo., pp. viii. + 358, with 142 illustrations. Price: $7.00 net.

results. Radium therapy and deep X-ray therapy, though admittedly in the experimental stage at present, receive no mention.

On the whole it cannot be said that this book comes up to expectations. While it may be of some value to the surgeon who performs prostatectomy occasionally or general practitioner who is not *au fait* with modern urological technique, it has nothing to recommend it as a book of reference to the expert in this department of work.

BIOCHEMICAL VARIATIONS OF THE NUCLEO-PLASMIC RATIO.

THE recent monograph by Le Breton and Schaeffer on the "Biochemical Variations of the Nucleo-Plasmic Ratio"[1] forms a valuable and timely contribution to knowledge of this important problem. The morphological variations of the ratio of nuclear to cell volume have attracted the earnest attention of biologists ever since R. Hertwig, in 1903, suggested that this ratio played a determinative part in fixing the moment of cell-division and therefore exerted a regulative function in development. Hertwig's original assumption that this ratio is constant and distinctive of each species of animal has been proved untenable by the researches of Conklin and many others, but Conklin has made the very suggestive observation that at any given stage of development and in similar tissues the ratio of nucleus to cell volume is in fact constant. It is, therefore, necessary to amplify Hertwig's original hypothesis by the further supposition that each of the many physiological types of cells comprising the tissues of a higher animal is characterized by its own specific nucleo-plasmic ratio at division.

With two exceptions determinations of this ratio have hitherto been based solely upon measurements of volume. Now, as Conklin has pointed out, the nucleus contains several distinct morphological components, variously designated chromatin, linin, nuclear sap and so forth, which are not present in the nucleus in the same proportions at different periods in the life-cycle of the cell. During division large quantities of nuclear sap are taken up from the surrounding cytoplasm and this disappears or is consumed during the growth of the cell prior to the next succeeding division. Mere measurement of nuclear volume, therefore, gives a very inaccurate idea of the quantity of the most distinctive nuclear component, chromatin, and an approximation to accuracy can only be achieved by carrying out the measurements, as Conklin did, at like periods in the life-cycle of the cells.

The exceptions to which reference has been made are those afforded by the estimations of Masing and Shackell on the changes of nucleic acid content of embryos (estimated in terms of phosphoric acid) during development. The many inaccuracies which vitiated these estimations, now twelve years old, are carefully pointed out by Le Breton and Schaeffer who conclude that chemical estimations of this ratio have not hitherto been sufficiently accurate to afford any safe basis for generalization.

Le Breton and Schaeffer have devised a new method of determining the alteration of the nuclear content of the embryo during development which depends upon the estimation of the amino-purines, adenine and guanine. The chemical technique which they employ, is very thoroughly controlled by estimations in solutions of known concentration and the recovery of known amounts of guanine and adenine after their addition to tissue.

They propose to employ the ratio of amino-purine nitrogen to total nitrogen after subtraction of the amino-purine nitrogen from the latter as a measure of the nucleo-plasmic ratio. The possibility that nucleic acid might in part be derivable from extra-nuclear sources is not discussed. The values of the ratio thus estimated fall off continuously during the development of the chick, pig and mouse, thus confirming the more recent estimates of the morphologists. They attribute the diminution of the ratio to the accumulation of non-active materials (paraplasm), an inference which involves the supposition for which no proof has as yet been advanced, that the ratio of nuclear materials to active protoplasm is constant throughout development.

[1] "Variations Biochimiques du Rapport Nucléoplasmatique au Cours du Développement Embryonnaire: Recherches de Physiologie Général sur la Détermination de la Masse Protoplasmique Active," par Éliane le Breton et Georges Schaeffer, Strasbourg; 1923. Paris: Masson et Compagnie; Royal 8vo., paper cover, pp. 196, with 16 figures. Price: Frs. 15 net.

PERSONALITY AND ENDOCRINE GLANDS.

THE researches in psychology and in endocrine functions are gradually bringing about an interesting situation; for two schools of writers are arising, one of which ascribes all human ills to psychical changes and the other to endocrine function or disfunction. Dr. Louis Berman belongs to the latter school and in his work entitled "The Glands Regulating Personality," he seeks to develope the thesis that all human personalities depend on the quality and quantity of their internal secretions.[1] "All the different nuances of personality are expressions of a particular relationship between the endocrines and the viscera and muscles." With the knowledge we have already gained of the very close relation between various aspects of mentality and certain endocrine secretions, this is a perfectly fair proposition to discuss. But if the attempt be made, the subject should be discussed scientifically and in the cold light of reason. Unfortunately the writer does not do this. Judging from the introduction and from the plan of the work, he writes rather for the information of the general public than of the medical profession. There is a fairly accurate historical résumé of the gradual increase in knowledge concerning the endocrine glands. This is followed by chapters dealing with their activities. The main theme of the work is then developed and analyses of certain historic personages are added. As the book is intended for the general public, scientific language is not to be expected, but there is all the greater need for accuracy of statement and cleanness in writing. The ordinary reader has no means of testing accuracy on such a subject. But the writer of this book apparently is one of those who thinks that a thing need only be repeated often enough and dogmatically enough to make it true. He draws no distinction between what has been scientifically proved and what has been tentatively suggested. This is particularly true of his account of gland activities. Pages are full of such phrases as "might be," "possibly" or "there is reason to believe" and so long as they fit in with his theory, such statements have equal weight in his mind with proved facts.

He is possessed of a fertile imagination which he uses to illustrate his theories. But he is as careless in his use of language as in his use of facts. In three successive paragraphs, he states man is the "creature" of his glands, man is "regulated" by these glands and man is "dominated" by such glands, as if creation, regulation and domination were one and the same thing. His fertility of imagination and language leads to a mixture of metaphors and a flow of words that is alternately amazing and amusing. In the first two pages of the chapter on "The Glands as an Interlocking Directorate" (whatever that may be) he writes of the body-mind being a perfect corporation of which the glands are the "directorate." Then he writes of two directorates; "infections may cause reverberations in both directorates." But what is a reverberation in a directorate? In the next paragraph, the control is called a board of governors, then a "House of Glands," then an "invisible committee behind the throne." Darwin has been accused of many things, but this writer says he "changed fate from a static sphinx into a chameleon flux." Some of these mixed metaphors are priceless. "Why does the slave soul infiltrate like a cancer the soul of society with its black fluid?" "The little men ploughed their little fields, striking the oil of a great generalization with no fanfare of trumpets." "To the Freudians we owe the aeroplanes to a new

[1] "The Glands Relating Personality," by Louis Berman, M.D.; 1921. London: Macmillan & Company, Limited; New York: The Macmillan Company; Demy 8vo., pp. 300. Price: 18s. net.

universe. They have opened up the geology of the soul and what a melodramatic cinema they have unfolded." And who with any sense of humour could write such a sentence as the following? "We seem to be much nearer to grasping the nature of the unconscious, when we look upon it as a historical continuum, a compound or emulsion of different and various states of intravisceral pressure and tone, in the vegetative apparatus, dependent upon the balance between the endocrines, as well as upon past experiences of the viscera in the way of stimulation or depression." This certainly brings us very near to the unconscious state.

With such a vocabulary, it is regrettable that the author should feel compelled to invent words like "unsloughable" and "tyrannosaurus," or indulge in phrases like "there is another and a rarer bird, the careerist of talent," or "more rot is talked." What is a "slave-beast brute"? What is meant by "egoisms of alimentation"?

Inaccuracy of statement, extravagance of thought and absence of perspective become supreme in the endocrine analyses of certain historic personages. According to this writer, Darwin "infected himself with the virus of neurasthenia"—during his voyage on the *Beagle* of all places. His whole life is explained on the grounds of a "hyperfunctioning pituitary, the anterior portion dominating the posterior, a thyroid excess and an adrenal much deficient." His adrenals sentenced him to destruction; he survived as a genius so long "because his wife and children supplied him with the endocrine energy he lacked"!

Napoleon is classed as a "pituitary centred, ante pituitary superior, post pituitary inferior with an instability of both that would lead to his final degeneration." His irritable bladder, of course, was due to pituitary disturbance, even though *post mortem* examination revealed stones in the bladder. In the same way the careers of Julius Cæsar, Oscar Wilde, Nietzsche and Florence Nightingale are all quite simply explained!

An excellent jest. But the concluding chapter on applications and possibilities is alarming. We are to have a science of puericulture by means of endocrine analysis and vocational education according to the results of these tests, the personnel of industries being chosen accordingly. Worse than all "the public health officer of the future will be armed with a new weapon in his fight against the spread of an epidemic. He will be able to classify the endocrine traits of the population exposed and to advise a course of glandular feeding for the types specially liable." For (according to this author), "diphtheria occurs most virulently among 'adrenal poor' individuals; the thyroid dominant rarely if ever suffers from measles; the pituitary type is more liable to epidemic meningitis and infantile paralysis, typhoid and scarlet fever; the mortality of the next influenza epidemic is to be lowered by the 'proper' handling, from the endocrine standpoint, of the thymo-centrics and the related adreno-centrics."

There is only one word to describe all this and we are debarred from using it, because we have already objected to its use by the author.

NURSING THE INSANE.

THE long-looked for seventh edition[1] of the "Handbook for Attendants on the Insane" has now appeared under the new name of "Handbook for Mental Nurses." The sixth edition of this handbook was issued as far back as December, 1911, but no less than six reprints have been called for in the intervals.

This edition which has indeed "made broad its phylacteries and enlarged the borders of its garments," has been entirely rewritten: the scope of the book has been enlarged and follows closely the syllabus of training for mental nurses laid down by the Medico-Psychological Association. It now contains thirteen sections: from an introduction on the general duties of mental nurses, to a long dissertation on mental deficiency, that *bon bouche* of psychiatry which is so frequently dished up now-a-days to tickle the palate of the public. It is not quite clear why this wide and rather incohate subject of mental deficiency should have been dragged in, by the hair of its head as it were, to a book designed for the training of mental nurses. Especially does a doubt of its utility arise when the arguments for and against Binet's and Simon's and other tests are discussed. A book designed for the instruction of, presumably, only moderately educated pupils, should not confuse the issues by the introduction of discussions of alternative methods, but should confine itself to positive statements. However, the fault of this edition is that it repeats, nay rather accentuates, the fault that was evident in previous editions, namely the inclusion of too minute details of anatomy and physiology, especially that of the nervous system, for a work designed for the instruction of members of the nursing staffs of mental hospitals whose general education usually falls very far short of a high school standard, whereas the text given on the finer anatomy and physiology of the brain (for example the physiological levels) would require the best attention of a fourth year medical student for adequate assimilation. The authors, indeed, seem aware of this elaboration of detail and say such detail "is essential for a thorough understanding of the work" and also "adds interest to the reader's studies." The trouble is that the ordinary nursing probationer, not fortified by previous education, is very unlikely to assimilate such "strong food," but will nevertheless try to master it, to the neglect probably of much more important, though less interesting, practical aphorisms; with the result that a bad attack of mental indigestion produces only a hopeless confusion of mind.

The general get-up, printing, type and paper are excellent and the greater number of the subjects have been adequately dealt with. Under the heading of "Water Supply" there might well have been given an enumeration of water-borne diseases and the likely contaminations of drinking water. The classification of forms of mental disease formerly given is now omitted, possibly because of the difficulty of arriving at a completely satisfactory classification. Teachers usually find it useful to have a simple table to set before their classes. Under the description of "Insane Habits and Propensities" more detailed methods of dealing with or counteracting these bad habits might have been given with advantage.

On the whole, despite the drawbacks mentioned, this book will prove distinctly useful in the training of nurses in hospitals for the insane.

[1] "Handbook for Mental Nurses," published under the Authority of the Medico-Psychological Association: Seventh Edition; 1923. London: Bailliere, Tindall & Cox; Demy 8vo., pp. xiv. + 640, with two plates and nineteen figures in the text. Price: 6s. net.

THE FORM AND FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM.

DR. Tilney and Dr. Riley have published a second edition of their book entitled "The Form and Functions of the Central Nervous System." The first edition of this work was favourably reviewed by us in October, 1921. We spoke of it then as being different from any other book we had read, inasmuch as the anatomy and physiology of the nervous system, instead of being allowed to stand independently, were incorporat d as essential parts of the practical knowledge needful to the proper diagnosis and treatment of disease. In other words, under one cover, the author has produced an integrated exposition of anatomical, physiological and clinical neurology, an arrangement which greatly facilitates study.

In the new edition there are no material alterations in the text or illustrations, the only changes are that some improvements suggested by reviewers have been made and some typographical errors corrected.

We again commend the work and at the same time congratulate the writers on its favourable reception.

[1] "The Form and Functions of the Central Nervous System," by Frederick Tilney, M.D., Ph.D. and Henry Alsop Riley, A.M., M.D.; Foreword by George S. Huntington, Sc.D., M.D., Columbia University; Second Edition; 1923. New York: Paul B. Hoeber; Crown 4to., pp. xxvi. + 1019, with 591 figures, containing 763 illustrations of which 56 are coloured. Price: $12.00.

The Medical Journal of Australia

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The Medical Congress.

Recently the Council of the New South Wales Branch of the British Medical Association entertained the members of the Federal Committee at dinner at the Australian Club. It fell to the Chairman, Mr. G. A. Syme, who was designated the leader of the medical profession in Australia, to respond to the toast of the evening. Part of his speech was devoted to the subject of the first session of the Australasian Medical Congress (British Medical Association), of which he is President. Mr. Syme had much to say about the work of organization and about the intellectual feast that the office bearers of the twelve sections are preparing for those members who will be wise enough to attend the Congress. In his enthusiasm to convince his audience that the preparations were well in hand and that the sectional programmes were no longer skeletonic, Mr. Syme risked his reputation as a bright speaker. But even if the members, content and in a comfortable frame of mind after an elegant and sumptuous repast, were scarcely inclined to give rapt attention to details of scientific programmes, Mr. Syme undoubtedly attained his objective. Everyone was impressed with the competence and earnestness of the workers in Melbourne. Having told his story concerning the spade work carried out at head-quarters, Mr. Syme attacked the other end of the problem. It should be remembered that the success of an intellectual effort like that of organizing a scientific congress depends almost as much on the manner in which advantage is taken of the facilities provided by the organizers as on the efficiency of the organizers themselves. It would be futile to expend energy and money, to display ingenuity and resource and to enlist the enthusiasm of a large army of helpers unless the same enthusiasm were spread throughout the six States of the Commonwealth. Mr. Syme referred almost pathetically to the thoughtless habit of members of leaving it to the last moment before they decided whether or no they would attend. This necessarily leads to increased difficulties for the Executive Committee, both in imposing a strain on the office during the last few days when members rush to join and in rendering it impossible for them to arrange for the intellectual and social entertainment of all.

The first session of the Congress is to be held in Melbourne which is topographically the most accessible city in Australia. Under the new organization the medical profession will be given a better congress than they have had hitherto. The march of science and the growth of the medical profession combine with the improved machinery to produce this effect. Each and every member of the British Medical Association owes it to himself and to this great body representing the profession to make a special effort to free himself for the week. If he has a message to give to his colleagues, he has no right to absent himself. But whether he has something of importance to say or not, he should recognize that by staying away, he will miss a special opportunity of extending his knowledge and of widening his views. Few can afford to waste such an opportunity. It is often urged that we in Australia suffer from the disability of not being able to meet experts in the many branches of scientific knowledge for the interchange of ideas. The European certainly has this advantage. Just because this opportunity is relatively rare in the Commonwealth it should be grasped with eagerness and avidity. The British Medical Association has been asked to arrange for the attendance of its President to celebrate the inauguration of the Australasian Medical Congress (British Medical Association). Sir William Macewen, the President for the year 1922-1923, has accepted this invitation. Mr. Syme pleaded for an earlier application for membership. We would extend his pleading and ask every member of the British Medical Association to make a special effort to avail himself of this great opportunity and to notify without further delay the honorary secretary in the State in which he resides, that he intends to be present at the Melbourne session.

Current Comment.

TETANY.

THE production of experimental tetany in animals by the removal of the parathyreoid glands led to the belief that tetany in human beings, whether occurring in adults or children, was due to a deficiency of the internal secretion of this gland. On account of the similarity of the clinical manifestations of the condition many observers including Macleod have assigned to infantile convulsions and *laryngismus stridulus* the same probable origin. This is, however, by no means proven. MacCallum and Voegtlin suggested that tetany might be due to a loss of calcium from the tissues on account of the relief caused in the symptoms by the administration of calcium. As a result of later experiments they concluded that the action of calcium was due to its depressing action on the motor centres. MacCallum also found that tetany could be relieved by the administration of sodium chloride. Howland and Marriott maintained that a lowered calcium content of the blood was responsible for what is known as idiopathic tetany. They based their conclusions on observations made on the blood of children suffering from the disease. Paton did not deny that any relationship existed between the calcium ions and the symptoms of tetany, but he ascribed the latter to intoxication by guanidine. He showed that the administration of guanidine to normal animals produced symptoms similar to those produced by parathyreoidectomy. He held that no drug other than guanidine had been found that was able to produce an increase in the excitability of the motor nerve endings to the constant current. He found an increase of guanidine in the blood and urine of dogs that had been submitted to parathyreoidectomy and in the urine of children suffering from tetany. He found that the serum of dogs from whom the parathyreoid glands had been removed, acted on the muscles of the frog in a manner similar to weak solutions of guanidine. He also found a similarity in the relative amounts of the nitrogenous metabolites in the urine of dogs from whom the parathyreoid glands had been removed and in the urine of normal animals injected with solutions of guanidine.

Dr. Wilder Tileston and Dr. Frank P. Underhill have recently investigated the condition of three adult patients suffering from tetany in regard to alkalosis and calcium metabolism.[1] The first patient was a woman, aged thirty-five years, who was suffering from Weil's disease with acute nephritis and acidosis. Large amounts of sodium bicarbonate were administered to her, with a diet poor in calcium. After nine days of such treatment she developed tetany and the carbon dioxide combining power of the blood rose from twenty-one to eighty volumes per hundred. The second patient was a woman, aged forty-six years, who developed tetany after influenza. She had had two previous attacks of tetany. The carbon dioxide combining power of the blood was seventy-seven per hundred and the calcium content of the serum estimated during a latent period was normal. The third woman, aged thirty-five years, had suffered from fatty diarrhœa for six months. The cause of the diarrhœa was not ascertained. Attacks of tetany were frequent. The sodium bicarbonate content of the blood was high and that of calcium was low. Indican was present in the urine. The patient died after an illness of five months' duration. No *post mortem* examination was made. The increased sodium bicarbonate content of the blood in the first patient was attributed to the administration of the drug. In the other two the observers were not able to assign a cause for the finding. They say that it is important to note that an increased carbon dioxide combining power of the blood does not necessarily mean a change in the hydrogen-ion concentration toward the alkaline side, as the increase in bicarbonate can be and in most instances probably is compensated, so that no change in the reaction of the blood takes place. They hold that the term alkalosis should be restricted in use to cases in which the hydrogen ion concentration of the blood is actually increased. They discuss their findings in regard to calcium in these three patients and refer to the work of many others on calcium metabolism in this regard. They conclude that some forms of tetany may occur without any change in the calcium concentration in the blood and that decrease of calcium is not of itself enough to cause tetany.

The whole subject is extremely fascinating and as Drs. Tileston and Underhill state there is much yet to be learned of the pathogenesis and ultimate causation. Quite possibly Macleod is right in his statement that it is likely that neither guanidine nor calcium deficiency is the primary cause of tetany, but that they may be secondary to some cause not yet discovered.

[1] *The American Journal of the Medical Sciences*, May, 1923.

PLEURAL REFLEX.

LAST year Dr. L. S. T. Burrell and Dr. A. Salusbury Macnalty published a special report in the Medical Research Council series on artificial pneumothorax. A full summary of this report was published in the issue of this journal of November 4, 1922. In dealing with the dangers of pleural puncture and the injection of gas into the pleural cavity these authors gave the first place to pleural shock, but maintain that this is a rare accident. They encountered it but twice in the course of 2,332 punctures. They ascribed the symptoms to a reflex induced by the undue stimulation of the pleura and recommended the injection of morphine or "Omnopon" and the local use of "Novocain" as the best means of preventing it. Many observers have reported their experience of the pleural shock or reflex and several hypotheses have been set up to explain its mechanism. The majority have recognized that the cardio-respiratory failure, the tonic and clonic convulsions and the loss of consciousness following immediately the introduction of the needle

into the chest constitute a reflex phenomenon of some kind. Dr. Barnett P. Stivelman has attacked this problem from a broad point of view.[1] From the published records it would appear that in the practice of some clinicians the pleural reflex is not as uncommon as Drs. Burrell and Macnalty would lead the reader to suppose. In his own sanatorium Dr. Stivelman encountered the symptom complex thirteen times in the course of 162 first punctures and 1,824 secondary punctures. In 1915 T. Sachs collected the experience of twenty-four American authors who had performed thoracentesis 1,122 times. The accident of this nature had occurred in the course of 134 punctures. In regard to the mechanism of its production, Dr. Stivelman excludes gas embolism, toxin absorption and similar causes on the ground that the reflex appears with such dramatic suddenness that there is no time for these changes to occur. It is more frequent after "dry taps" than after the injection of gas, so that gas embolism cannot be the cause. In his own experience pleural reflex occurs when the visceral pleura is injured in the attempt to inflate the cavity. In the majority of instances there was an extensive pleuro-pulmonary infiltration and fibrosis. He found that the reflex did not occur when the needle entered the widely open pleural cavity during the performance of refilling. He also noticed that when the reflex occurred, it was likely to recur in subsequent attempts to induce pneumothorax. He recognizes a cardio-inhibitory type in which the heart is slowed and the pulse is slow and forceful. Respiration is inhibited in this form. This type is rarely fatal. The other type, the vaso-motor, is much more dangerous. It is characterized by a steady depreciation of the pulse and a failure of the respiration. At times the two types coexist. He holds that the best method of preventing the accident is to employ short needles, not exceeding three centimetres in length, with a narrow bore. Great care should be taken in the selection of the site of puncture. The clinical as well as the radiographic evidence of the condition of the pleura and lung should be carefully studied and recourse to the needle should be withheld save when it is certain that the two layers of the pleura are not adherent. If there is evidence of inflammation of the visceral layer of the pleura and of the lung immediately covered by the inflamed membrane, the puncture should be made as far away from the site of inflammation as possible, if it is carried out at all. Under no circumstances should the needle be moved about within the cavity.* It is much safer to withdraw the needle and to introduce it several times in different situations when the first puncture is unsuccessful. It would appear that Dr. Stivelman's explanation would hold good for nearly all the recorded pleural reflex eccidents. The surprising variation in the frequency of the accident in the different clinics and hospitals can readily be explained on the assumption of a varying degree of caution in introducing the needle in the presence of signs of localized pleuritis. The clinician who refrains from using the needle for diagnostic purposes and who depends on the results of clinicial examination and on what the skiagram has to divulge, is less likely to injure the visceral layer of the pleura and the underlying lung than he who pays little attention to physical signs. Attention should be paid to the experience gained in sanatoria and hospitals in regard to the signs elicited when repeated attempts to produce an artificial pneumothorax prove resultless. Drs. Burrell and Macnalty have some very wholesome lessons to give in this regard. The value of this procedure need not be disregarded on account of this grave accident. It is preventible and the best preventive is extreme caution and a search for clinical signs of inflamed pleura before the puncture is attempted.

[1] *The American Journal of the Medical Sciences*, June, 1923.

COMPLEMENT FIXATION IN LEPROSY.

THE serum of lepers, as has long been recognized, often yields a reaction to the Wassermann test. An investigation of this fact in regard to leprosy has recently been made by Dr. E. W. Goodpasture.[1] Dr. Goodpasture tested the sera of three groups of patients. The first group consisted of thirteen untreated patients known to have leprosy. The second group consisted of fourteen who had received injections of chaulmoogra oil but from whose blood the *Bacillus lepræ* could still be isolated. The third group consisted of sixteen patients who had been treated and were apparently free from the bacillus of leprosy. A reaction was obtained in the sera of eight of the first group and in eleven of the second group. Of the sera of the last group none yielded a reaction. Dr. Goodpasture states that the small percentage of untreated reacting leprous patients and the possible presence of infection by syphilis or yaws are important considerations in eliminating it as an indication of the progress of the patient towards recovery from leprosy. He turned his attention to bacterial antigens and has found more promising results from the use of suspensions of the *Bacillus tuberculosis* (human type). When the relation of these acid-fast bacilli to one another is considered this is not surprising. A strong reaction occurred in the complement fixation test with this antigen in every one of twenty-four sera from patients suffering from leprosy and just beginning treatment. Of twenty patients with leprosy who gave no clinical or bacteriological signs of the disease as a result of treatment, four yielded a strong reaction, three failed to react and the remainder showed a definitely weakened reaction. These results are regarded as indicating the possibility of applying the test to leprous patients as a means of measuring their response to treatment. Although the tubercle bacillus is not the causal organism in this disease, a specificity of the antigen is not essential in view of the fact that patients with known infections are being tested. Dr. Goodpasture states that when a strong reaction persists after a clinical "cure" of the leprosy, the presence of a complicating tuberculous focus should be suspected.

[1] *The Philippine Journal of Science*, April, 1923.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Specificity of Mesothelium.

R. S. CUNNINGHAM (*Bulletin of the Johns Hopkins Hospital*, July, 1922) has studied the changes in the omentum of the rabbit during mild irritations, with especial reference to the specificity of the mesothelium. The omenta in regard to which the author has reported his observations, were taken from a series of rabbits whose peritoneal lining cells had been irritated by the injection of heterogeneous laked blood. Some of these rabbits were vitally stained by the intravenous administration of trypan blue and a few received the vital dye intraperitoneally in order to give the irritated serosal lining cells every possible opportunity to absorb and store the dye. The author found that the serosal lining cells and the fibroblasts react differently, both as regards their morphological appearance and the distribution of their vital dye content. The serosal lining cells increase in thickness and are much more compact than the normal, flat, plate-like structures. On the other hand, the fibroblast during irritation becomes an elaborately branched structure, forming a marked contrast to the compact serosal cell. In the serosal cell the vital dye content is more diffuse than in the normal cell, but there is never any dye to be seen in any of the fine processes. In the fibroblast the vital dye is distributed throughout the processes, the granules often being collected in the bulb-like extremities. It is thus obvious that during the course of a mild irritation the changes which take place in the mesothelial and fibroblastic elements indicate that they become even more widely separated from each other in their group characteristics. It seems entirely justifiable to conclude that such differences must denote some variation in the structure of the cytoplasm and hence indicate some especial adaptation for certain definite physiological activities. In addition to these morphological criteria, it has been demonstrated that the vital dye content of the fibroblast and of the mesothelial cell presents a sharp contrast. That differences in the way in which vital dyes are stored, represent corresponding differences in physiological adaptation seems most probable and permits further security to be placed in the conclusion that the mesothelial and fibroblastic elements are wholly specific.

Oats in Stomach Wall.

T. SHENNAN (*The Journal of Pathology and Bacteriology*, January, 1923) reports an instance of foreign body tubercles on the serous coat of the stomach, caused by the escape of particles of oatseed. The patient, a married woman, thirty-six years of age, gave a history of indigestion of several months' duration. She complained of more or less constant discomfort across the upper part of the abdomen, aggravated by food. On the day before admission to hospital she had an acute attack of pain. Laparotomy revealed no sign of acute inflammation, ulceration or new growth; but an eruption of raised pink nodules—in size up to that of a grain of boiled rice—was seen over the visceral peritoneum in the right upper quadrant and mainly over the pyloric end of the stomach. These were at first thought to be tuberculous, but in consistence they differed from tuberculous nodules and their distribution and position were unusual. On microscopical examination the nodules were found to be made up of granulation of two types. The first type consisted of young, vascular, œdematous connective tissue, almost myxomatous in character, which contained fibroblasts and a little delicate fibrillar intercellular material, with occasional large and small mononuclear cells and a few polymorpho-nuclear leucocytes. The second type consisted of more cellular areas in which were embedded elongated fragments of a vegetable substance, of which the largest measured one millimetre in length. Closely applied to the surfaces of these particles were numerous foreign body giant cells. Along with the giant cells were many endothelioid cells accompanied by polymorpho-nuclear leucocytes and lymphocyte-like cells. Young oval and elongated connective tissue cells were scattered throughout and the whole cellular area was enclosed by concentrically arranged, loose, fibrous connective tissue. Plasma cells were conspicuous by their absence. The particles contained in the nodules were demonstrated to have come from grains of oats. It is assumed that the particles escaped through a small perforation in the stomach wall which healed up speedily and perfectly without much reaction even locally.

Differences Between Mitochondria and Bacteria.

E. V. COWDRY AND P. K. OLITSKY (*Journal of Experimental Medicine*, November 1, 1922) state that mitochondria show microchemical and tinctorial properties differing from those of bacteria. In respect to mitochondria, spores and capsules have not been noted. Motility due to flagellar action has not been observed and there are certain differences in staining reaction. The temperature, oxygen and food requirements of mitochondria can only be expressed in terms of the requirements necessary for the vitality of the cells as a whole. No mitochondria are known to resist a temperature of over 50° C.. Neither is there any parallelism between mitochondria and bacteria in their relation to fermentation and to disease. In fact, mitochondria tend to decrease rather than to increase in diseases of infective nature. Furthermore, there is no reason to believe that mitochondria possess the power of independent and characteristic growth apart from cells. The suggestion that mitochondria are independent micro-organisms rests in the author's judgement upon no other evidence than a similarly in form of substances about the same size.

Coal Tar and the Liver.

J. DAVIDSON (*The Journal of Pathology and Bacteriology*, January, 1923) applied coal tar to the external surfaces of both ears of rabbits and found that different degrees of change were produced in the liver. In the acute type death occurred after nineteen days with three applications of tar. Macroscopically the liver showed large, pale, yellow areas interspersed between paler red areas; small petechal hæmorrhages were also present. Microscopically there was comparatively little fatty change. Numbers of the cells showed vacuoles which did not contain fat. Other areas showed an acute necrosis with infiltration of small round mononucleated cells and polymorpho-nuclear leucocytes. Other animals showed similar changes of a sub-acute or chronic nature. The livers of full-time young whose mothers had been treated with tar, showed extreme vacuolation and healthy parenchymal cells were difficult to find. These experimental conditions are comparable with acute yellow atrophy and atrophic cirrhosis.

Polyposis of the Stomach and Duodenum with Carcinoma.

H. H. GLEAVE (*The Journal of Pathology and Bacteriology*, January, 1923) reports an instance of polyposis of the stomach and duodenum with carcinoma in a female, thirty-six years of age. At autopsy the stomach showed a large annular ulcer on the posterior and superior walls. The floor of the ulcer was partly formed by the pancreas. Numerous polypi were present (about fifty), varying from small sessile nodules on the mucosa to well defined polypi, usually attached by narrow stalks. The largest measured 7.5 centimetres (three inches) in length by 2.5 centimetres at its greatest diameter. In the pylorus and duodenum polypi were present. The intestines showed nothing of note except one small polypus in the rectum. The liver contained innumerable deposits of new growth. This is the second case of polyposis of the stomach seen in the last six thousand *post mortem* examinations at the Leeds General Infirmary.

MORPHOLOGY.

The Lymphatics of the Uterus.

J. NEVEUF AND H. GODARD (*Revue de Chirurgie*, Number 3, 1923) record the results of four years' work on the lymphatic drainage of the uterus. The method employed was to inject selected sites of the uterus with one injection only of Prussian blue solution and then to determine the distribution of the dye in the various lymphatic channels. The uteri of about one hundred and fifty newly-born children were employed and the results were confirmed in uteri of

two young adults. They found that both for the cervix and the body there is on each side a common main path which they name "the principal channel" and several "collateral or accessory channels." The "principal channel" is formed by a convergence of the lymphatics of the body and the cervix to the borders of the uterus just above the utero-vaginal junction. From this point a number of lymph vessels follow the uterine artery in front of the ureter as far as the outer third of the broad ligament where they pass outwards and slightly forwards and become associated with the obliterated umbilical artery. Then either directly or after passing deeply they enter a gland or ganglion —"the principal ganglion"—situated about fifteen millimetres in front of the bifurcation of the common iliac artery and either lying on the external iliac vein or more frequently below the vein upon the obturator nerve. The efferent vessels of this gland or ganglion cross the external iliac vessels and ascend to the outer side of the common iliac vessels, the aorta and *vena cava*. They then pass beneath the great vessels on each side, join and enter the *cisterna chyli*. The "collateral or accessory channels" of the cervix consist of two retro-ureteral channels. One group passes in company with uterine veins to the side wall of the pelvis to one of the glands lying among the terminal branches of the internal iliac artery. From this gland efferent vessels arise which pass to the inner side of the common iliac vessels towards the promontory of the sacrum. This path they name the "hypogastric channel." The second group medial to the former follows the upper border of the hypogastric sympathetic nerves as far as the promontory, where it enters a gland in relation to the left common iliac vein. This is the "sympathetic channel." The efferent vessels of these two groups unite to form a single channel which lies to the inner side of the common iliac vessels on each side. Above the promontory they pass outwards, cross the common iliac vessels and join the corresponding "principal channel." The "accessory channel" for the body of the uterus is important. Commencing at the uterine cornua the lymph vessels traverse the upper part of the broad ligament, pass with the ovarian vessels across the iliac fossa to the lower pole of the kidney where they turn inwards anterior to the uterers to enter glands in front of the aorta and *vena cava*. The efferent vessels of these glands unite with "the principal channel" behind the aorta before entering the *cisternia chyli*. In no instance did any of the injections made in the usual course enter the lymphatics of the round ligament.

Gigantism and the Pituitary Body.

E. UHLENHUTH describes further experiments upon the feeding of the anterior lobe of the hypophysis to salamanders (*Journal of Experimental Zoology*, January 5, 1923). Metamorphosed salamanders of the species *Amblystorma tigrinum* were fed on earthworms, on anterior lobe of the pituitary body taken from cattle and upon ox liver. Both the pituitary and liver fed animals grew more rapidly than those fed on earthworms. The effect of the pituitary body was, however, to maintain growth for a larger period and also the animals reached a much greater size than any of the others. Hence anterior lobe of the hypophysis, among the food substances so far tested by the author, produces the largest animals. This excess of size may be as much as 25.4% in excess of the size of the largest known normal animal of this species. The largest liver-fed animal exceeds the normal maximum size of the species by only 5%. An important feature was that since the age limit for growth was reached in none of the animals examined, it would seem that the cessation of growth was determined by the size to which a given substance can make the animals grow.

Growth and Variability in the Norway Rat.

HELEN DEAN KING (*The Anatomical Record*, March 20, 1923) outlines the results of investigations upon the growth and variability in the body weight of the Norway rat as compared with the domesticated albino rat. The animals studied were chiefly trapped round Philadelphia and litters were reared in the laboratory. A total of one hundred and ten offspring were reared up to as long as two years. They for the most part resisted attempts at taming, especially after weaning. Besides recording a large mass of data, certain prominent features are discussed. In the first place, Norway rats do not show the definite acceleration in body growth during early life that is characteristic of the albino strain. Here the author remarks that rapid growth during early life seems to be characteristic of most domestic animals and that graphs of growth of Norway wild rats under domestication from the second to the fifth generation, show progressive approximation to those of the albino rat. While for a short period the females grow more rapidly than the males, after the sixty-day period the males show a constantly greater rate of growth and also both sexes tend to increase in weight throughout adult life. The Norway rat shows a high degree of variability in body weight at all ages and the males, except in early life, show a greater variability than the females. Variability in body weight is seemingly correlated both with rate of growth and with age in the rat and in man.

Germinal Centres in the Spleen.

J. S. LATTA (*The Anatomical Record*, November 20, 1922) continues his investigations on the germinal centres in the lymphatic tissue of the spleen. He first gives a short historical review in which he draws attention to the looseness with which the term "germinal centre" has been used and shows that the term was originally used by Flemming (1885) to indicate the lighter central portion of the nodules. Flemming considered these areas as centres of proliferation of lymphocytes. Later writers have frequently used the term to indicate the complete lymphatic nodule. The material studied by the author consisted of fresh human spleens collected at autopsy and spleens of dogs, cats, guinea-pigs and rabbits. An account of the histological appearances is appended and some attention is given to the presence of large acidophilic macrophages, which are to be seen more especially in these lighter coloured germinal centres. While in agreement with other observers as to the presence of mitotic figures in the outer dark staining ring of these lymphoid nodules the author considered the lighter central areas or "germinal centres" as areas of degeneration and adduces evidence in support of his hypothesis.

Cystic Artery.

G. L. MCWHORTER (*Surgery, Gynecology and Obstetrics*, February, 1923), in discussing a new method of gall bladder dissection, draws attention to its surgical anatomy. Four relations between the cystic artery and bile ducts may be considered normal. In the first and most frequent form the right hepatic artery with normal origin passes posteriorly to the bile ducts and gives off the cystic artery to their right. Less frequently the right hepatic artery passes anteriorly before giving off the cystic branch. The cystic artery may arise to the left of the common bile duct and pass either anteriorly or posteriorly to it. Anomalies of the cystic artery may be either its unusual origin, number or course. In nine out of twelve instances in which the origin of the cystic artery lay to the left of the bile ducts from some hepatic branch, it passed anteriorly to the common bile duct and the right hepatic artery in view of the circumstances passed posteriorly. In twenty-six instances of the same series in which the cystic artery arose to the right of the common bile duct, the artery giving rise to this branch passed anteriorly to the bile ducts in five instances and posteriorly in twenty-one. In twenty of these instances the artery giving off the cystic branch was the only branch to the right lobe of the liver. In two instances the right hepatic artery lay in close relationship to the cystic duct. In four instances the cystic artery arose from a right hepatic artery and had its origin from the superior mesenteric artery. In one instance it arose from an accessory right hepatic artery arising directly from the aorta. The course of the cystic artery, hepatic artery or one of its branches lay to the right of the common or cystic ducts and parallel to the right free edge of the lesser omentum in one instance in about every seven examined. In one instance in every twelve the cystic artery was found to be double. In three instances they both arose from the right hepatic artery or the gastro-duodenal artery; or one arose from the right and one from the left hepatic artery.

Special Abstract.

DIPHTHERIA, SCARLATINA AND IMMUNITY.

SURGEON-COMMANDER SHELDON F. DUDLEY, O.B.E., Professor of Clinical Pathology and Lecturer on Tropical Diseases in the Medical School of the Royal Naval College at Greenwich, engaged some time ago in an investigation with the the object of ascertaining whether the Schick test for immunity to diphtheria might be of use in conserving the fighting efficiency of the Royal Navy. This investigation resolved itself into an elaborate and well-planned study of the conditions obtaining at the Royal Naval School at which there are about one thousand boys in residence and from one hundred to two hundred day boys. The result of his work and of that of some of his colleagues is now available in a report of the Medical Research Council.[1] The information contained in the report is of undoubted importance and Surgeon-Commander Dudley's ingenuity in evolving a very plausible hypothesis in regard to the occurrence of immunity among the community will command immediate attention. It is unfortunate that he lacks the power of clear exposition and that he, like many others working in immunology, should have had recourse to unnecessary abbreviations and the use of contradictory terms. In the following summary, an attempt will be made to reproduce his facts and deductions in unambiguous terms. More particularly the confusion between cases and patients and between the results of test and reactions will be avoided.

Immunity to Diphtheria.

The author attacks the problem by recording the incidence of diphtheria in the school and by inquiring into the way in which the boys responded to the Schick test. Some of the boys at the school at the time of the investigation had joined in September, 1917. From that date until January, 1919, there were only one or two isolated infections in the school. From January to May, 1919, no less than sixty-five boys were admitted to the infirmary wards on account of diphtheria. No further outbreak occurred for two years. During the term from May to July, 1921, twenty boys were ill with diphtheria. The author points out that it appears from the record that some of the eighty-five boys did not actually suffer from acute diphtheria, but were merely diphtheria bacilli carriers. Since he has no definite evidence that the bacilli found in the fauces of these boys were Klebs-Löffler bacilli, he assumes that they must be regarded as bacilli morphologically indistinguishable from true diphtheria bacilli. He goes out of his way to introduce the symbol "M.D." for these bacilli. Between September and December, 1921, one hundred and three boys were infected. In addition two boys had typical membranous diphtheria, but no *Bacillus diphtheriæ* could be found. Of these one hundred and five boys, sixty-three had membranous diphtheria, fourteen had non-membranous sore throats from which diphtheria bacilli were isolated and twenty-eight were bacillus carriers without clinical manifestations of the disease. Some of them had scarlatina. Between January and April, 1922, fourteen boys had diphtheria and between May and August, 1922, thirteen boys had the disease. It thus appears that between May, 1921, and August, 1922, one hundred and twelve boys were found to be suffering from diphtheria judged from a clinical point of view and a total of one hundred and fifty-two boys harboured bacilli morphologically indistinguishable from diphtheria bacilli in their throats. During the same period over one hundred and fifty boys suffered from scarlatina. All these boys were boarders, not a single day boy having been infected.

The Schick test was applied to seven hundred and sixty-four old boys and eighty-six new boys, making eight hundred in all. These boys were grouped into eleven companies. The largest company comprised one hundred and sixteen boys. The boys of the several companies mixed indiscriminately at work, at play and meals. They were grouped in their dormitories. The results of the test are of three kinds. In the first place there is the reaction, by which it is inferred that the individual boy was susceptible to diphtheria. In the second place a so-called pseudo-reaction was noted. This is a reaction to the heated fluid in which the toxin is destroyed. The heated fluid is used as a guide on the "control" arm. The third result is a failure to react, held to indicate an immunity to diphtheria. In some instances a reaction may be associated with a pseudo-reaction. The boys yielding such a response would be susceptible to the disease. Those who gave neither a reaction nor a pseudo-reaction, are grouped as immune. Those who yielded a pseudo-reaction, but not a true reaction, are classed as non-immune. The author introduces a complicating element by the use of the illogical and contradictory terms "positive reactors," "negative reactors," "pseudo-reactors." Moreover, he employs symbols instead of terms in the most unnecessary manner. Of the old boys only 14% gave a reaction, while 24% gave a pseudo-reaction either combined with a true reaction or not. No less than 45% of the new boys yielded a reaction. This means than 86% of the old boys and 55% of the new boys were said to be immune. Similarly there were 9% of the new boys giving a pseudo-reaction.

Of the one hundred and twelve boys who had at one time or another harboured diphtheria bacilli, 86% were immune and 14% reacted. Of the fifty boys who had had membranous diphtheria, 84% were immune and 16% reacted. Of one hundred and nine boys who had had scarlet fever, 82% were immune and 18% reacted. Of sixty-six boys who had had non-specific sore throat, 85% were immune and 15% reacted. Of six hundred and fifty-two boys whose history did not reveal an infection with diphtheria bacilli, 86% were immune and 14% reacted. In connexion with the boys who had had clinical diphtheria, the author records that three months later no less than 92% failed to respond to the test, that is had become immune. The facts ascertained in this initial investigation impelled the author to study the development of immunity among the boys with extreme care. The new boys were three times as susceptible as the old boys. Two attacks of diphtheria or infection with bacilli in the same boy did not occur in the school. On inquiry he found that immunity to diptheria, as expressed by a failure to react to the Schick test, increased with length of residence in the school. The increase, however, was not gradual, but proceeded in sudden spurts. Of the boys who had been at school during the two epidemics, 95% were immune; of those who had been at school during only one epidemic, 84% were immune. Of the boys who had been at school for periods not exceeding one term, only 65% were immune. He gives evidence to show that the development of the immunity is not a mere matter of age. He also adduces strong reasons in favour of the view that a manifest infection is not necessary for the development of immunity. Further applications of the test revealed that 32% of a series of one hundred boys who reacted to the test, became immune in the course of three months. It was noted that a pseudo-reaction appeared together with a true reaction in twelve of these boys. After three months only one maintained the same response; three yielded a true but not a pseudo-reaction, seven yielded a pseudo-reaction but not a true reaction and one did not react at all. In no circumstances was the change from immunity to susceptibility. The question of the significance of the pseudo-reaction is also attacked. The author regards this response as being caused by some thermostable substance in the broth culture used for the test other than the toxin. After a consideration of all the evidence available, he arrives at the conclusion that the pseudo-reaction is often a step on the way to immunity. It probably signifies a relatively recent sensitization to the proteins of the diphtheria bacillus. With care it is possible to eliminate doubt in the great majority of the individuals yielding a pseudo-reaction.

During the period from September to December, 1921, swabs were taken of 1,250 boys. Bacteriological examination demonstrated that 4.7% of these boys were carriers of bacilli morphologically indistinguishable from diphtheria bacilli, while 7% suffered from membranous diphtheria. Some cultures were sent to the Lister Institute for examination and it was found that all but one were toxin-forming Klebs-Löffler bacilli. This matter was investigated on a

[1] "The Schick Test, Diphtheria and Scarlet Fever: A Study in Epidemiology," by Surgeon-Commander Sheldon F. Dudley, R.N.; Special Report Series, No. 75, Medical Research Council of the Privy Council, London, 1923.

larger scale. Bacilli from unselected and from selected carriers and from boys suffering from membranous diphtheria were examined. Of twenty-two strains, fifty-two proved to be virulent and twenty avirulent. True diphtheria bacilli were recovered from 95% of the boys with clinical diphtheria; 49% of the bacilli from the carriers were virulent.

The author was able to study the response to the Schick test in fourteen boys who were later attacked with diphtheria. In thirteen there was a reaction. One boy failed to react and his throat harboured virulent bacilli. If the condition from which this patient was suffering was true diphtheria, there must have been something very unsual in the circumstance. The author examined the condition carefully and arrived at the conclusion that the disease was diphtheria. The boy had occupied the bed next to one used by a boy who proved to be a dangerous bacillus carrier. The history of this boy showed that he infected others very readily. Surgeon-Commander Dudley suggests that the patient who had failed to react to the Schick test, was not sufficiently immune to withstand the massive doses of bacilli emanating from this carrier. Later he encountered three other boys who failed to react to the test and who were attacked by a condition regarded to be diphtheria. Careful revision of the diagnosis, however, showed that the disease was not diphtheria.

In the next place he inquired into the immunity response of carriers. Eleven boys were discovered who were harbouring virulent diphtheria bacilli in their throats. None of these reacted. Twelve other boys carried avirulent bacilli in their throats. Of these four reacted to the test. The author maintains that carriers of virulent bacilli who react to the Schick test, are unknown. One boy who had reacted ten weeks before and who subsequently was found to be a carrier of virulent bacilli, failed to respond when the test was repeated a fortnight later. Of the four carriers of avirulent bacilli, two were retested and then it was discovered that they no longer reacted. The third was attacked by clinical diphtheria shortly after. This seemed to indicate that avirulent bacilli do not influence the patient's immunity to the disease.

The Incidence of Diphtheria and Scarlatina.

During the term September to December, 1921, there was a heavy morbidity rate of scarlet fever, diphtheria and sore throat. By sore throat is meant an illness characterized by faucial inflammation without rash and without diphtheria bacilli being found in the faucial mucus. During this triple outbreak there were one hundred and five boys with diphtheria, one hundred and twenty-three with scarlet fever and sixty-six with sore throat. The two hundred and ninety-four boys affected represented 29.4% of the school population. Curves of the incidence reveal that that of diphtheria was longer and flatter than that of scarlet fever. The sore throat curve follows the scarlet fever curve more closely than it does the diphtheria curve. Surgeon-Commander Dudley examined the epidemiology of these infections. He points out that while diphtheria bacilli carriers were detected and isolated, it is impossible to recognize the carrier of the infecting agent of scarlet fever. An examination of the distribution of the beds occupied by these patients was carried out. While the affected boys were distributed throughout all the dormitories, it was found that when two or three boys were attacked together, they usually slept in the same dormitory. The beds are arranged in the dormitories in three rows of beds placed head to foot. The space between the beds is one and three-quarter feet (53.5 centimetres), while the passage between the set of three beds is five feet (152 centimetres). The ventilation is ample and the cubic air space generous. It was found that in one dormitory nineteen boys were infected with scarlet fever during the term and seven were infected with sore throat. All but two of the boys with scarlet fever slept in one half of the dormitory and of these seventeen twelve were in adjacent beds. In those instances when two boys in adjoining beds were affected, the interval between the onset of the fever was less than the maximum incubation period of scarlet fever. The distribution of the boys with sore throat does not support the suggestion that this illness was a larval form of scarlet fever. In four of the seven, however, the boys occupied beds adjacent to the beds with boys who were infected with scarlet fever.

Inquiry failed to elicit a bed to bed spread of diphtheria either from patient to patient or from carrier to patient. Full information is not available and the author points out that he is unfortunately unable to ascertain which boys were immune as judged by the response to the Schick test and he is also of opinion that all the carriers may not have been detected, since a systematic examination of every boy in an adjacent bed to the patient was not carried out immediately on the discovery of the infection.

No connexion was made out between infection with diphtheria bacilli and infection with Hoffmann's bacilli.

In concluding this chapter on the epidemiology of these infections, Surgeon-Commander Dudley recalls the work of Glover, who demonstrated that the most important factor in the spread of the meningococci from soldier to soldier was the distance between the men's beds. The administrative rule is that a safe distance of at least two feet, six inches (76 centimetres) should be maintained.

Methods of Spread of Infection.

The author again refers to the singular fact that not a single infection occurred among the day boys. He argues that this must indicate that the majority of infections take place in the dormitories. His inquiries support this view, especially in regard to scarlet fever. In order to examine this question, the author found it necessary to consider the general grouping of the boys in the school. The school is divided into companies, messes and classes. He therefore endows each boy with three numbers to indicate his position in regard to this division. A table setting forth the total number of boys in each company, in each class and in each mess and also the number of boys infected with diphtheria and with scarlet fever under each heading reveals no obvious difference between the distribution of the two diseases, which would suggest that they are spread by the same means and in the same way. In five classes of senior boys the average morbidity rate for scarlet fever was 19.2%, while for diphtheria it was 17.6%. The average morbidity rates for the remaining fifteen classes were 10% and 7.2%. From the table it appears that it was rare for two boys from different dormitories, but in the same class or mess, to fall ill with the same disease on the same day. Three boys from the same mess but from different dormitories, were attacked by scarlet fever on the same day, but as the disease was very prevalent at the time throughout the school, this occurrence was likely to take place. It appears further that before September 8, 1921, there had been no diphtheria for a considerable time. Within a week from that date five boys from five different dormitories, five different classes and five different messes were taken ill with the disease. After an interval of twenty days three other boys were infected. Two of these boys came from the same class. After a further twenty-three days another boy was taken ill with diphtheria from a different company and a different mess. This company contained seventy boys, who played, worked and ate with boys from other companies with infected members. After the infection had occurred, 20% of the boys in the company became infected. The same set of circumstances was discovered in regard to scarlet fever. In order to find a common source of infection that led to the almost simultaneous outbreaks of diphtheria and scarlet fever in individual boys belonging to five separate dormitories, much thought was expended and Surgeon-Commander S. Roach made an important discovery. It was found that a certain boy was a heavy carrier of diphtheria bacilli. This boy was in the habit of sucking his pen-holder. At the end of the lesson he, following the custom prevailing, threw the pen-holder into the common class-room pen box. Another class would use the same room and the boys of this class would take up pen-holders from this box. The carrier probably infected pen-holders in each class room in which his class had lessons. It was noted that the day boys supplied their own pen-holders and had private pen boxes.

Direct experiments demonstrated that a pen-holder that had been sucked by a bacillus carrier, sufficed to give rise to a culture of diphtheria bacilli in broth, even after it had been stored in a cupboard for fourteen days. At the

end of six weeks no bacilli were recovered. The author has satisfied himself that this mode of infection explains the escape of the day boys and also the spread of the infection from one dormitory to another.

The Diagnosis of Diphtheria.

The diagnosis of diphtheria should be based on the presence of the characteristic pseudo-membrane, on the reaction to the Schick test and on the isolation of virulent diphtheria bacilli. At times it becomes necessary to determine whether or not diphtheria is present when one or other of these three characteristics is absent. The author emphasizes the importance of the clinical evidence and maintains that no good clinician will wait for the result of the bacteriological examination or of the Schick test before administering antitoxin. He goes one step further. He states that even if no bacilli are found and the Schick test does not yield a reaction, antitoxin must be given unless diphtheria can be excluded on clinical grounds. He holds that there is no doubt that the absence of mortality among the boys with diphtheria was largely due to the promptness with which treatment was instituted. In doubtful cases the finding of virulent bacilli and the reaction on the part of the patient to the Schick test would confirm the diagnosis, while if no reaction to the Schick test were obtained, the conclusion would be justified that the patient was a bacillus carrier with some coincident infection. In the course of the investigations it was found that bacilli indistinguishable from true diphtheria bacilli were recovered from the throats of over 95% of the boys suffering from clinical diphtheria and that 95% of these bacilli were true, virulent diphtheria bacilli. The author attributes the failure in 5% partly to errors in clinical diagnosis and partly to faults in the laboratory technique. When a bacillus emanating from the throat of a diphtheria patient is recognized on morphological grounds as indistinguishable from diphtheria bacillus, it is highly probable that it is a true diphtheria bacillus. On the other hand, when a bacillus is recovered from the throat of a carrier, it is necessary to test its toxity before the definite diagnosis can be made.

Velocity of Infection.

The author, profiting from his experience at the Royal Naval School, has evolved an ingenious hypothesis to explain the spread of infection in a school or other community. He finds that the most usual mode of infection in the school is from boy to boy in the dormitories where the beds are placed relatively close to one another. He suggests that in all infectious diseases there is a certain minimal dose of infective agent necessary to cause the disease. If an individual receives a dose of the infective agent less than the minimum infective dose, it is destroyed by the defensive mechanisms of the body. He is inclined to believe that the infecting dose varies with every host and with every parasite, but that the limits of variation are definite for susceptible persons. When a person receives a succession of sub-minimal doses, the result must depend on whether there is sufficient time between the doses for the body to deal with the bacteria. He has encountered instances on ships and elsewhere in which the infecting dose has been fractional, that is sub-minimal doses repeated at short intervals. He assumes that the body can deal with a given amount of infecting agent in a unit of time. For the sake of clarity of thought he fixes the unit of time arbitrarily at one hour. If the velocity of infection is greater than the velocity of destruction of the infecting agent, infection will take place, provided that the process of infection continues for a sufficiently long time. On the other hand if the velocity of destruction is greater than the velocity of infection, no disease will result. In order for the disease to develope the patient will require to be exposed to infection for $\frac{\text{M.I.D.}}{\text{V—U}}$ hours, when M.I.D. equals the minimal infective dose, V the velocity of infection and U the velocity of destruction of the infective agent. In the school, when this period of time was more than one hour but less than ten, the absence of boy to boy infection in the class room and at meals and the heavy incidence of infection in the dormitories would be explained. This hypothesis would also explain why the day boys escaped both diphtheria and scarlatina infections while three hundred of the boys with whom they played, worked and ate, became infected with one or other of these infections. He points out that the infected droplets of saliva may be carried in the air as far as nine hundred or one thousand centimetres, yet coughing children can be treated in a ward in beds from 270 to 360 centimetres apart without any cross infection occurring. He argues that the absence of cross infection must be due to the low concentration of infection at 360 centimetres, so that the individual boy will not receive in one hour more infecting agent than his body can destroy. The likelihood of infection diminishes rapidly with the distance between the boys. In the dormitories the distance between the boys in bed was approximately 135 centimetres. At 270 centimetres the concentration would be one-eighth of the concentration at 135 centimetres and at 360 centimetres it would be one-nineteenth. Glover found that by increasing the distance between the beds of soldiers from 90 centimetres to 150, the carrier rate for meningococci diminished from 28% to 6%. This difference has approximately the same ratio as that of the cubes of the distances, that is five to one. The author therefore assumes that by doubling the distance between the beds at the school the velocity of infection would be diminished from V—U to $\frac{\text{V}}{8}$ — — U.

Surgeon-Commander Dudley next draws attention to the fact that some carriers are obviously more dangerous than others. The bacilli in the throat of the more dangerous carrier are not more virulent than those in the throat of a less dangerous carrier. During the influenza epidemic it was found that picture theatres and other places of amusement were more dangerous than trains. A man travelling for two periods of half an hour daily spent more time in the crowded environment than he did in attending once a week a performance lasting two or three hours. But the velocity of infection might be sufficient to produce an attack in the three hours of continuous exposure, but not sufficient to produce an infection during the thirty minutes in the train. He cites many other observations which reveal that the length of exposure to infection is an important ætiological factor. Bad ventilation must be regarded as a contributory agent. Infected droplets are carried farther in humid air and their concentration becomes greater in these conditions than when the air is moving well.

Immunity to Diphtheria and Antitoxin.

The author states that it is reasonable to assume that one of the chief factors in acquiring immunity to diphtheria is the appearance of antitoxin in the blood. This antitoxin would be produced by the stimulus of diphtheria toxin, the product of virulent bacilli which have either infected the individual or been absorbed by him. Certain facts, however, seem to oppose this hypothesis. In the first place, 85% of individuals are born with antitoxin derived from the mother. This antitoxin disappears, but the majority of persons acquire antitoxin at a later period. The number of those who react to the Schick test, increases in proportion to age at a greater rate than can be accounted for by previous attacks of diphtheria. In the next place, he finds that, even if the diphtheria morbidity were high enough to account for the immunity of those who fail to react to the Schick test, it would be necessary to explain how it is that when the effect of antitoxin given therapentically has worn off, so many patients after recovery from diphtheria again react to the test. Thirdly he finds that carrier infection cannot explain the acquired immunity to diphtheria, because the carrier who reacts to the test, does not exist or is extremely rare. His studies in the school show that many boys acquire antitoxin without clinical or bacteriological evidence of diphtheria, but they acquire this immunity only when there are ample chances of receiving diphtheria infection. He finds that nearly every boy became immune within nine months following the attack of diphtheria and that no boy was attacked a second time, although all were exposed to infection. He therefore claims that the two first objections are not valid. He argues that failure to react to the Schick test necessarily means that the individual has been in contact with diphtheria bacilli at one time or another. He is inclined to discard the doctrine that antitoxin in the blood or tissues is an essential part of immunity. Other-

wise it would be difficult to explain the immunity of the carrier who harbours bacilli that are capable of infecting others, but he himself does not react to the Schick test. The acquired immunity probably depends on the number, size and separation in time of the sub-infective doses of diphtheria bacilli. If the amount of infection received exceeds a certain amount within a given time, the recipient who fails to react to the Schick test, becomes a carrier. Or if he reacts to the test, he becomes infected. He recogizes that the question of the amount of infecting agent is important. He maintains that some boys react to the test, but are sufficiently immune to resist ordinary doses of diphtheria bacilli. He refers to the work of Glenny and Allen which tends to show that the toxin injected for the purpose of performing the Schick test may act as a secondary stimulus to the production of antitoxin. There is, however, evidence that natural antitoxin slowly disappears from animals who have been infected with diphtheria, but even after it has gone, the animal is still actively immune.

Epidemic Immunity.

The author takes his arguments a little further. He points out that large sections of the community with which he has been dealing, became immune to diphtheria without suffering from an attack during the presence of an epidemic of diphtheria. He therefore arrives at the conclusion that coincident with an epidemic of diphtheria, there is an epidemic of immunization. There was a remarkable similarity in the onset and distribution of scarlet fever and of diphtheria. Scarlet fever spreads more rapidly at first than diphtheria. In the coincident epidemics from September to December, 1921, there were one hundred and forty-five cases of scarlet fever and one hundred and fifty-two of diphtheria. He asks whether it should be assumed that the eight hundred and fifty boys who did not get scarlet fever, were immune at the beginning of the epidemic. Natural immunity to scarlet fever is held to be small as compared with natural immunity to diphtheria. Scarlet fever spread more rapidly because the concentration of susceptible individuals was greater. This more rapid infection induced a greater concentration of infection and consequently a more rapid dissemination of sub-maximal dose of infecting agent. These doses would immunize a proportion of the boys without any appearance of the clinical manifestations of the disease. In December, 1921, when the number of boys infected with scarlet fever was the same as the number of boys infected with diphtheria, it was probable that the number of the immune to scarlet fever was approaching that of the immune to diphtheria. This was found to be about seven hundred. During the following term the infecting agents of both diseases were present in the school and the opportunities for contact were identical. The concentration of susceptible individuals and of the centres of infection must have been the same for both diseases, since fourteen boys were attacked by scarlet fever and the same number were attacked by diphtheria. He offers this hypothesis of epidemics of infection and coincident epidemics of immunity as a more plausible explanation of the facts than the vague supposition of a mysterious change either in the virus or in the population.

British Medical Association News.

MEDICO-POLITICAL.

Transactions of Council of the Victorian Branch.

THE following is a summary of the more important transactions of the Council of the Victorian Branch of the British Medical Association during the preceding six months.

Friendly Societies.

The last of the friendly societies, the Manchester Unity Independent Order of Oddfellows, has accepted the terms offered by the Council of the Branch. At the request of the Manchester Unity a conference was held and the Order agreed to accept the terms of the Wasley Award together with certain provisions accepted by the other societies. There was an additional condition that the same charge must be made to all members of the Order whether they selected an institute or a non-institute doctor. To make the terms more readily acceptable to the general meeting of the Manchester Unity held at Sale, the Council agreed that no amendment of the Wasley Award would be made before the end of 1925. The general meeting accepted the provisions *in toto* and agreements have now been signed with the former lodge medical officers as from June, 1923. At the conference the Manchester Unity Independent Order of Oddfellows agreed to pay all accounts contracted by its members as private patients of members of the Victorian Branch of the British Medical Association during the period of the dispute; and the members of the Victorian Branch were asked by circular to send their accounts to the local secretaries for endorsement and submission to the central executive of the Manchester Unity Independent Order of Oddfellows.

Intermediate Hospitals.

The State Treasurer intimated to the Council his intention of introducing an Intermediate Hospitals Bill during the current session of Parliament and asked for any advice the Council might wish to offer. The Legislative Committee met on many occasions and a series of resolutions adopted by it was agreed to by the Council. At present the Committee is engaged upon a revision of the standard scale of fees. The principles adopted will be submitted to a special meeting of the Branch before being forwarded to the State Treasurer.

Bush Nursing.

It was frequently reported to the Council that bush nurses were transcending their limitations and were affecting to give medical service in a district where a medical man was engaged in active practice. A conference was held on April 19, 1923, with the central executive of the Bush Nursing Association and it was agreed to insist on the nurses observing the rules of the Association and to provide that where a medical practitioner is resident in a town, the bush nurse will attend only maternity and emergency cases without reference to a medical practitioner.

Reorganization of the Work of the Council.

For a long time it has been felt that a large amount of the work of the Council could be effected by its committees and by this arrangement the number of meetings of the Council could be lessened. A committee was appointed to inquire into this matter and its determinations were adopted by the Council and submitted to a special meeting of the Branch in the form of an amendment of the Rules. The Council will now meet on the third Wednesday in each month instead of twice a month as formerly. More powers will be given to subcommittees of the Council which will meet on specified dates and will support their deliberations to the Council for confirmation.

Coroners' Courts.

A letter was forwarded to the State Attorney-General calling attention to the unfairness of the coroners or deputy coroners making comments on the conduct of a medical practitioner without giving such practitioner the opportunity of rebutting such statements. The Attorney-General has expressed his disapproval of such procedures through the columns of the daily press.

Congress Ball.

The Council decided that the Branch should entertain visiting members of the Congress at a ball to be held in the St. Kilda Town Hall on Friday, November 16, 1923, and that the President of the Branch should act as host. This resolution was confirmed by a general meeting of the Branch.

War Memorial.

The memorial raised by members of the Branch in memory of the medical officers who fell at the war will be completed and unveiled during Congress week. The

Council of the University has given permission for the memorial to be housed in the new Anatomical Museum until such time as the Branch shall have been able to provide buildings of its own.

Health Officers' Salaries.

At the request of individual health officers, the Council has again actively urged the increase of salaries of health officers up to the rate recommended by the Public Health Commissioner. In some few cases favourable replies have been received.

Medical Officers of Education Department.

It was observed in an advertisement that the Education Department was calling for a medical officer with more than ordinary qualifications and hospital experience at a salary of £492 *per annum*. As similar positions in the Commonwealth Service carry a minimum of £606 rising to £798, with living allowances added, it was felt that a strong protest should be made against the inadequate salary offered. No appointment was made in the Department and a deputation waited upon the Minister, who thanked members for their support which would help him in his representations to the Cabinet.

New Buildings.

A committee of the Council has at times considered sites suitable for B.M.A. buildings, but found it would be necessary to approach the members of the Branch first before effecting the purchase of a site. A circular letter will shortly be issued asking to what extent members will financially support such a movement.

Homœopathy.

A movement for a *rapprochement* between members of the Victorian Branch of the British Medical Association and homœopathic practitioners was defeated in the Council. So long as such practitioners base their practice on an exclusive dogma, it was felt that no good could come of any consideration of the matter by the committee of the Council.

Locum Tenentes.

There has often come before the Council the question of *locum tententes* practising in the district where they have acted on behalf of the resident practitioner. The Council has held that such are debarred for all time from practising in that district without the consent of the principal concerned. It was resolved that *locum tenentes* when placing their names with the Medical Agency of the Medical Society of Victoria for employment should be warned of this disability. The Ethical Committee has been asked to draft such a circular and all medical agents in Victoria will be asked to distribute these to their clients on registering for employment.

Social Functions.

A dinner was tendered to Dr. S. W. Patterson on his departure for England. But he received an urgent cable to leave immediately and the dinner had to be abandoned. Dr. J. H. Anderson who also left for England, was entertained at afternoon tea and he was the recipient of a testimonial engrossed on parchment and a valuable presentation.

Representatives in England.

Dr. T. P. Dunhill was elected Representative of the Group on the Central Council. Dr. R. L. Wettenhall was elected Representative of the Victorian Branch on the Representative Body at Portsmouth and Drs. Gavin McCallum, W. J. Long and J. F. Mackeddie were appointed delegates.

Board of Masseurs.

At the request of the State Government the Council nominated two representatives on the Board of Masseurs, Drs. H. Murray and J. W. Springthorpe. These two have since been appointed.

Membership.

Subscriptions.

The Rules as to subscriptions were amended so as to provide that a member of not less than ten years' standing who has retired from active practice, or a member of forty years' standing shall pay two-thirds of the annual subscription.

Travelling.

Arrangements were made with the London office whereby members leaving Victoria temporarily could be placed on the "travelling list." Subscriptions will cease from the next half-year, the journals will be stopped and on the return of members to Victoria they will automatically resume full membership from the beginning of the last half-year.

Arrears.

Hitherto members whose subscriptions were two years in arrears, ceased to be members and had to be re-elected for membership. Such members are now reinstated upon the payment of all arrears. This course follows the new rule of the parent Association; but the Council reserves to itself that the ordinary formality of re-election should be complied with.

Election.

During the half-year fifty-one new members of the Branch have been elected by the Council.

THE Honorary Secretary of the Queensland Branch of the British Medical Association announces that there are some duplicate volumes of *The Lancet* in the Library of the Branch. These include the two half-yearly volumes bound separately from 1877 to 1901 and one or two earlier volumes. It is suggested that these volumes might be useful for the libraries of other Branches or that some members might wish to have them to complete existing sets. The majority are in good condition.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

DODSON, GEORGE HIRST, M.B., Ch.M., 1921 (Univ. Sydney), 43, Seymour Street, Croydon Park.

MONEY, REGINALD ANGEL, M.C., M.B., Ch.M., 1923 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

OLVER, LLOYD REATH, M.B., Ch.M., 1922 (Univ. Sydney), Victoria Avenue, Chatswood.

THE undermentioned has been elected a member of the Western Australian Branch of the British Medical Association:

O'BRIEN, MARK, L.R.C.P., L.R.C.S. (Edin.), 1896, L.F.P.S. (Glasg.), 1896, Perth.

HYDATID DISEASE.

DR. Harold R. Dew, Acting Director of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, informs us that a considerable amount of work has been carried out by the workers at the Institute in applying the immunological tests for hydatid disease during the past two years. While these tests have been performed primarily for the purpose of determining the exact value of the tests and of advancing knowledge in connexion with the pathology of this disease, the practitioners who have provided the material for the tests, have benefited considerably by the information obtained. Dr. Dew has recently circularized these practitioners, asking for information, but the response has been disappointing. Complement deviation, precipitin and Casoni tests have been performed with the blood of patients suspected to be suffering from hydatid disease. In many instances the

tests yielded results that justified the Institute workers to report that the diagnosis of hydatid disease was probably correct. Those who have received reports of this nature and who have not yet replied to the circular letter, are requested to inform Dr. Dew whether they have obtained any confirmatory evidence at operation or in any other way. Dr. Dew would further wish to receive details of the course of illness up to the time of reporting and also a fresh sample of the patient's blood. From six to ten cubic centimetres of blood are needed for the tests. We need scarcely point out that medical practitioners who are in a position to contribute to a research of much importance, should do so readily and willingly.

Post-Graduate Work.

SPECIAL POST-GRADUATE LECTURES IN MELBOURNE.

THE HONORARY SECRETARIES OF THE MELBOURNE PERMANENT COMMITTEE FOR POST-GRADUATE WORK announce that arrangements have been made with Professor C. J. Martin, F.R.S., for four lectures on "Disorders of Digestion" to be delivered in the Assembly Hall, Collins Street, Melbourne, on September 17, 18, 27 and 28, 1923. Tickets may be obtained from the Honorary Secretaries, Dr. J. W. Dunbar Hooper and Dr. Harold Dew (12, Collins Street, Melbourne). The cost of the tickets is two guineas.

Congress Notes.

THE PAN-PACIFIC SCIENCE CONGRESS.

THE following is the programme of the Sydney meeting of the Section of Hygiene of the Pan-Pacific Science Congress.

Friday, August 24, 1923.

Morning Session: 10 a.m. to 1 p.m.

Joint Meeting of all the Sections.

1. Inaugural Address by His Excellency the Governor of New South Wales.

Afternoon Session: 2.30 p.m. to 5 p.m.

1. Address by the Chairman, PROFESSOR C. J. MARTIN, C.M.G., F.R.S., on "Climate and Human Efficiency."

2. PROFESSOR SHOZO TODA: "The Natural Ventilation of Rooms of Various Construction; Experiments in Standardizing Labour of Dwelling Houses."

3. PROFESSOR SHOZO TODA: "Monthly Mortality-Rate Caused by Some Noteworthy Diseases Related to the Climatic Variation in Japan, 1913 to 1917."

Monday, August 27, 1923.

Morning Session: 10 a.m. to 1 p.m.

1. DR. C. E. CORLETTE: "On Some Relations of Climate, Weather and Fat Covering to Metabolism."

2. DR. H. S. H. WARDLAW: "Energy Consumption of Australian Students."

3. MR. BARKLEY: "Measurement of Climate."

Afternoon Session: 2.30 p.m. to 5 p.m.

Joint Meeting with the Section of Geography.

1. Discussion on Tropical Settlement.
2. PROFESSOR H. PRIESTLEY: "Physiological Observations in the Tropics."

Tuesday, August 28, 1923.

Morning Session: 10 a.m. to 1 p.m.

Joint Meeting of All the Sections.

1. Discussion on Settlement in Areas of Low Rainfall.

Wednesday, August 2, 1923.

Morning Session: 10 a.m. to 1 p.m.

Joint Meeting of All the Sections.

1. Discussion on Insects and their Rôle in Economic Problems of the Pacific.

Afternoon Session: 2.30 p.m. to 5 p.m.

Joint Meeting with the Sections of Entomology and Veterinary Science.

1. Discussion on Distribution of Insects in Relation to Disease.
2. DR. E. W. FERGUSON: "Distribution of Insects Capable of Bearing Disease in Eastern Australia."
3. DR. R. DICK: "Diseases Borne by Insects."

Thursday, August 30, 1923.

Morning Session: 10 a.m. to 1 p.m.

Joint Meeting of All the Sections.

1. Discussion on Climate, its Origin and Relations to Economic and Social Life.

Afternoon Session: 2.30 p.m. to 5 p.m.

1. DR. C. H. B. BRADLEY: "The Stegomyia Problem."
2. DR. J. PURDY: "Destruction of Insects."
3. DR. HARVEY SUTTON: "District Distribution of Disease Among Children in New South Wales."
4. PROFESSOR T. G. WELLS: "Heredity in Cancer" (communicated by PROFESSOR F. P. SANDES).

Friday, August 31, 1923.

Morning Session: 10 a.m. to 1 p.m.

Joint Meeting of All the Sections.

1. Discussion on the Organization of Research in the Pacific.

The following additional papers will be contributed at the Melbourne meeting.

1. PROFESSOR SHOZO TODA: "*Eine modifizierte Methode der Wohnungsdisinfektion vermittels Formaldehyd gegen die Verbreitung der Tuberkulose*" (A Modified Method of Disinfecting Houses Against the Spread of Tuberculosis).
2. DR. C. NANALING: "Hookworm Campaign in the Province of Cebu, Philippine Islands."
3. DR. C. NANALING: "Effect of Carbon Tetrachloride on Intestinal Parasites, Particularly the Hookworm."
3. DR. FRANK G. HAUGHWORT: "Industrial Importance of Intestinal Diseases."

Correspondence.

ERYTHRŒDEMA.

SIR: As the writer of the original article on Erythrœdema, I protest against the term "pink disease" being applied to it. It is quite true that I was not quite satisfied with the word, as there is no œdema in the general acceptance of this term, but œdema comes from οἴδημα which means swelling or tumour. There is very definite swelling, in fact the swelling with the redness are the most striking features of the disease and really made me try and find one word to describe it.

Dr. Littlejohn in his paper, never mentions any swelling at all, only emphasizes the fact that no œdema was present. I have never referred to the "pinkness of the hands and feet" as an œdema or "rash" or "erythematous eruption." I have never used the word pink as, in my opinion, it is entirely misleading and a very feeble designation. The distinguishing colour is not "pink": it is red or bluish red. No other observers use the word "pink" except Dr. Jeffreys Wood in his heading. Dr. Weston says: "Swollen, cold, bluish red." Dr. Field remarks: "The red swollen appearance of his hands and feet strike one immediately." Dr. Vipond: "The hands and feet look to be œdematous and the skin presents a bluish red colour." Dr. Thursfield: "The extremities were cyanosed, slightly œdematous and cold" and again: "Redness, cyanosis, desquamation."

One begins to wonder whether there is not a variant type of the disease in Sydney, seeing that the colour is different and there is neither swelling nor œdema. In the absence of these two signs, the word erythrœdema would never have entered my mind.

Dr. Field considers this name should be adhered to as it "so well expresses the most striking features of the disease, *viz.* the red swollen extremities." Dr. Parkes Weber thinks the name erythrœdema should be retained until more information as to the nature and ætiology of the condition is forthcoming. He also remarks with justice, that the name myxœdema is retained for a disease in which there is generally no true œdema.

Yours, etc.,

H. SWIFT.

Adelaide,
June 30, 1923.

THE COMPLEMENT FIXATION TEST IN HYDATID DISEASE.

SIR: In your issue of July 7, 1923, in the course of a review of Professor Hermann Kehl's monograph on "Surgical Diseases of the Abdomen Caused by Parasites," it is stated that "the author displays the narrowness of his perspective by attributing the complement fixation test to Weinberg. Hamilton Fairley's priority cannot be challenged."

In an article entitled "The Complement Fixation Test for Hydatid Disease and its Clinical Value" in THE MEDICAL JOURNAL OF AUSTRALIA, April 1, 1922, Hamilton Fairley reviews the more important literature dealing with this subject. He states that "Ghedini (1907) first applied the principles of the Bordet-Gengou method of serological diagnosis to three cases of hepatic hydatid. . . . Weinberg (1909) investigated in some detail the value of the serological reaction in human echinococcosis and published several papers on the subject."

It would appear that the priority of Ghedini's work is undoubted.

Were Dr. Hamilton Fairley in Australia, he would be the first to point out this error.

Yours, etc.,

K. DOUGLAS FA'RLEY.

Moonee Ponds, Victoria.
July 10, 1923.

"INSULIN."

SIR: Re the conditions attached to the use and distribution of "Insulin," as appears in the issue of THE MEDICAL JOURNAL OF AUSTRALIA, July 14, 1923, may I ask where one is to find "competent specialists" in Australia in the use of "Insulin"? Have Australian medical men to be classed in a lower grade of efficiency than their medical brethren in England? In England during the first few weeks when the supply of "Insulin" was limited, only certain hospitals were allowed supplies, but when the supply was equal to the demand, the use of "Insulin" was available to all medical men in England (*The British Medical Journal*, May 26, 1923). From the statement that appears in THE MEDICAL JOURNAL OF AUSTRALIA, it would seem that the use of Australian "Insulin" is to become a monopoly amongst a few medical men. Such a condition should be instantly removed. And yet, it hardly seems worth the trouble, in view of the fact that English-made "Insulin" is now procurable in some quantity at many places in Melbourne.

Yours, etc.,

J. M. BAXTER, M.D. (Melb.).

6, Collins Street, Melbourne,
July 17, 1923.

Medical Appointments.

DR. C. E. JELBART (B.M.A.) has been appointed Public Vaccinator at Cohuna, Victoria.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

AUG. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 15.—Victorian Branch, B.M.A.: Council.
AUG. 15.—Western Australian Branch, B.M.A.: Branch.
AUG. 16.—City Medical Association, New South Wales.
AUG. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 21.—Illawarra Suburbs Medical Association, New South Wales.
AUG. 24.—Queensland Branch, B.M.A.: Council.
AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
AUG. 30.—South Australian Branch, B.M.A.: Branch.
AUG. 31.—New South Wales Branch, B.M.A.: Branch.
SEP. 5.—Victorian Branch, B.M.A.: Branch.
SEP. 6—New South Wales Branch, B.M.A.: Nomination of two candidates to Federal Committee.
SEP. 7.—Queensland Branch, B.M.A.: Branch.
SEP. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
SEP. 12.—Western Australian Branch, B.M.A.: Council.
SEP. 12.—Melbourne Pædiatric Society.
SEP. 14.—New South Wales Branch, B.M.A.: Clinical Meeting.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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